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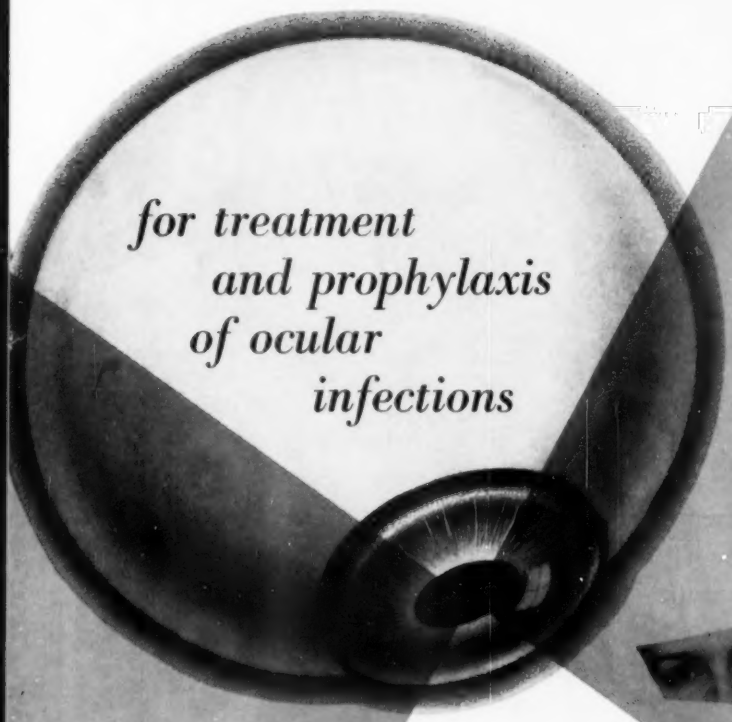

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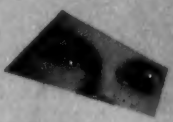

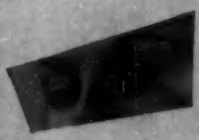

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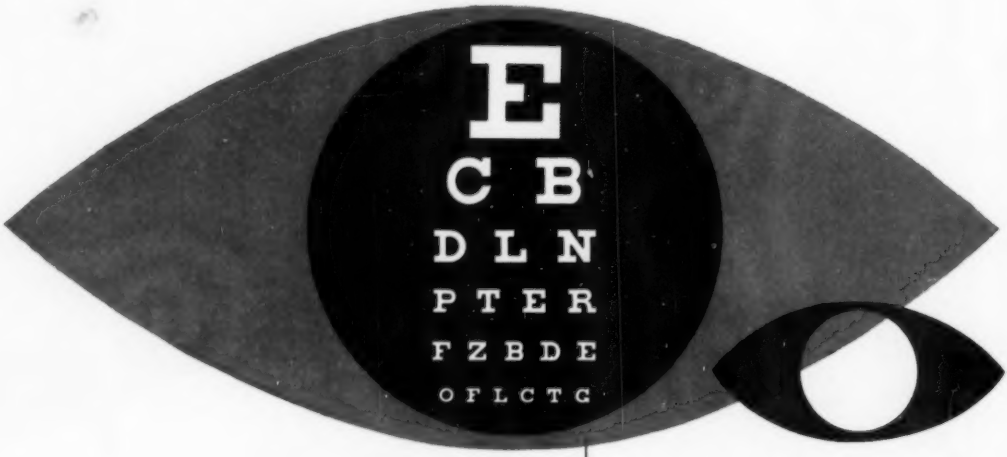

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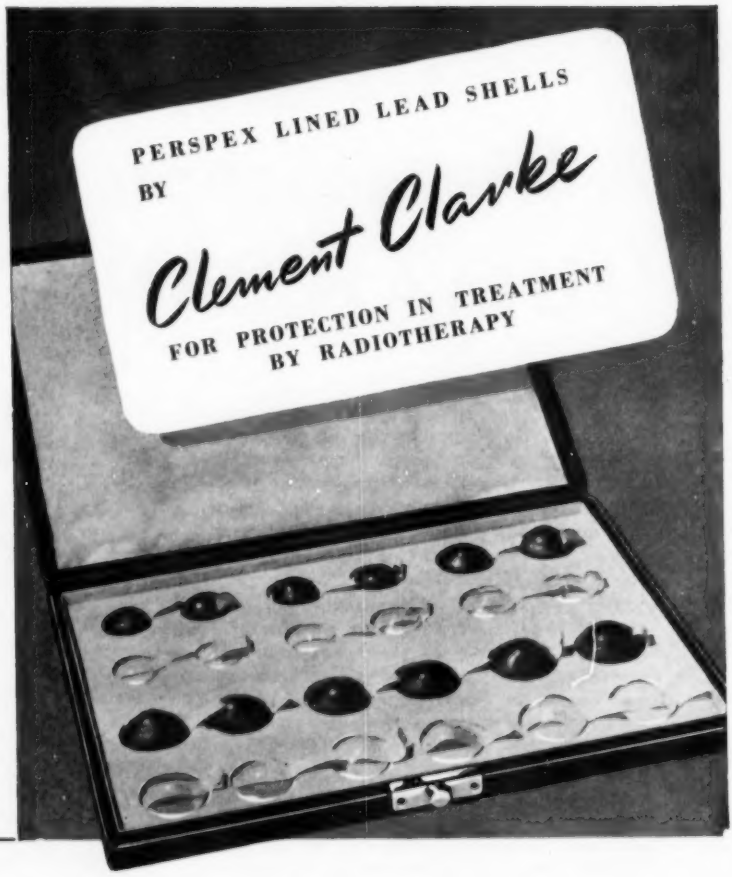

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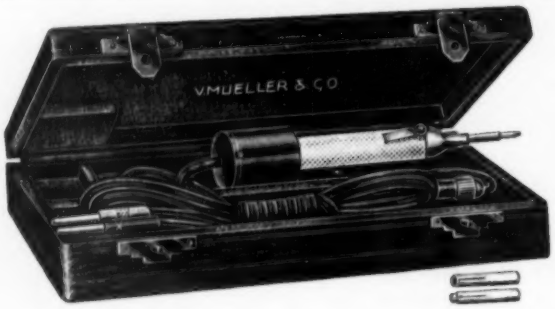

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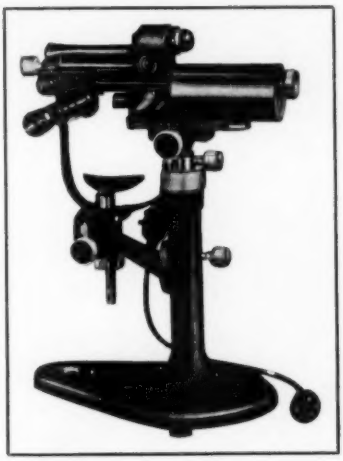

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VOLUME 52 DECEMBER 1954 NUMBER 6

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FREQUENCY OF RETINOBLASTOMA IN THE PROGENY OF PARENTS WHO HAVE SURVIVED THE DISEASE

ALGERNON B. REESE, M.D.
NEW YORK

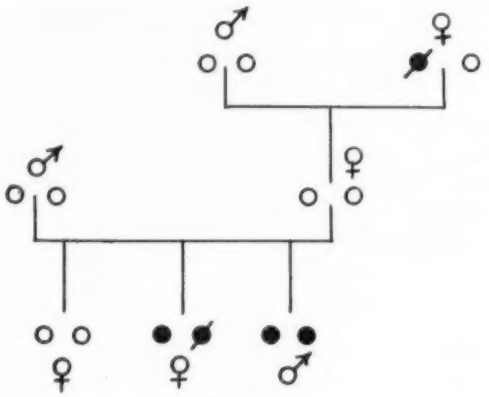
TWO QUESTIONS of genetic interest in relation to retinoblastoma are posed the ophthalmologist. First, when retinoblastoma has occurred in one child of healthy parents, what is the likelihood that further siblings will be affected? Second, is it advisable for the adult survivor of retinoblastoma to have children?

In an effort to answer these questions, in 1949 I [1] reported the results of an analysis made from the follow-up of 91 cases. For the purpose of this analysis, we communicated with 171 consecutive cases, and we were successful in receiving the desired data on 91.

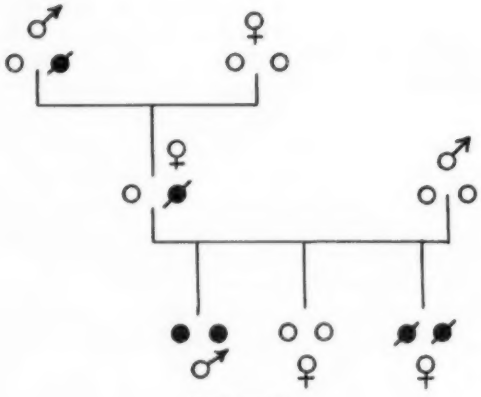
Eighty-six of our 91 cases were sporadic (no relative of the child having had the disease and the parents both having normal eyes), and 60 of these had siblings totaling 103. Only 1 sibling of these 103 had retinoblastoma. We could find in the literature only two other series (those of Reiser and of Hemmes) in which the total number of siblings was given. In determining the likelihood of the occurrence of retinoblastoma in siblings, we therefore had to base our deductions entirely on these three series, which together comprise 150 children, who had a total of 378 siblings. Among these siblings there was only 1 case of retinoblastoma, making the incidence less than 2.9 in 1,000. An analysis of the data in our series by the method of the confidence range indicates that if 1 of 103 siblings had retinoblastoma there is a 95% likelihood that the true range lies between 0 and 4% of siblings of patients with retinoblastoma born of healthy parents. The use of the total of 378 siblings in the three series would of course yield a considerably lower confidence range. An analysis, therefore, of several series of unselected cases of sporadic retinoblastoma indicates that the likelihood that a second sibling will be affected is less than 4% and is probably nearer 1%. I concluded, therefore, in this report that we should not hesitate to advise healthy parents who have had one child with retinoblastoma to have more children.

The frequency of direct transmission is more difficult to estimate, as there is no statistical analysis of any appreciable number of parents who have themselves survived retinoblastoma. Therefore, an accurate determination of the likelihood of transmission of the disease from a surviving parent to a child has not been possible. In our former report 5 of the 91 cases analyzed concerned patients who were the progeny of survivors of retinoblastoma. In these five families in which one parent had a verified retinoblastoma there was a total of nine children, eight of whom had retinoblastoma. Since then we have observed 10 additional families in which there was direct transmission. Their children totaled 21, and 15 had a retinoblastoma. Combining our total number of cases in the two series, we have 30 children whose

father or mother had confirmed retinoblastoma. Twenty-three children, or 77%, suffered from the tumor. Of the 15 families concerned in this analysis, there was only one in which all children, 3 in number, were free of the disease. It might be well to state that this one family out of the 15 was one not encountered in our routine observation of retinoblastoma cases. It has been my practice for years to investigate the children of any suspected survival. The one family free of the disease was



Pedigree 1

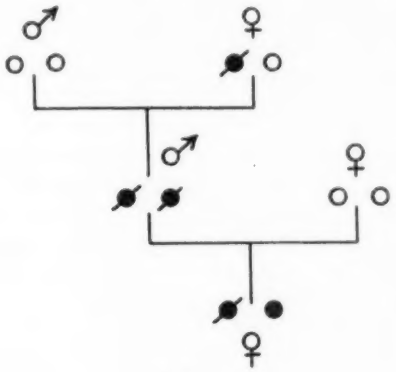


Pedigree 2

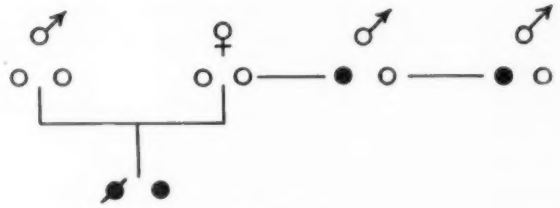
encountered in this way. A father brought his young child for some unimportant eye condition. Upon my questioning him regarding the cause of an enucleation which he had had, he told me that the removal was necessary because of a tumor of the eye in childhood. After communicating with the hospital where the enucleation was done, I was able to confirm the fact that the father had had a retinoblastoma. I examined his three children, who were 4 years old (twins) and 6 years old. With general anesthesia, I found the fundus of each eye of each child normal. This family, therefore, has been added to the series, although it was not encountered in a routine

consecutive manner. I should point out, though, that because of an especial interest in retinoblastoma it is possible that I would see a larger number of affected children of survivors, as those with healthy children would not have an occasion to seek medical help. This possibility, though, is somewhat balanced by the fact that ever since I can remember I have been on the alert for survivals with children.

In the former report, I concluded from the cases we had at hand that a survivor of retinoblastoma should not have children. The addition of our larger series certainly confirms this conclusion. Furthermore, an interesting problem from the genetic point of view is posed. If we assume that we are dealing with a dominant



Pedigree 3



Pedigree 4

gene, as was pointed out by Franceschetti,* theoretically, 50% of the children should be affected. In our series 77% were affected. The standard deviation (σ) is 3.52. Therefore, the difference between 77% and 50% could arise by chance in less than 0.1% of times. Also, chi square=8.54, indicating that the difference could occur by chance in 0.3% of the time. Thus, there is a definite statistical significance. Franceschetti † explains this difference on the hypothesis that the mutation appears most frequently in the retinoblastoma patient himself.

Several pedigrees of interest will be cited.

1. The maternal grandmother of two of our patients had her right eye removed in childhood for retinoblastoma (not confirmed by histologic examination). Both

* Franceschetti, A., cited by Dollfus and Auvert,[2] p. 34.

† Franceschetti, A., cited by Dollfus and Auvert,[2] p. 21.

parents of the patients have healthy eyes. Of their three children, though, two (a boy and a girl) have bilateral retinoblastoma and one has healthy eyes. This case was not used in our series here because the transmission was not direct; it skipped one generation (Pedigree 1 ‡).

2. We have two families in which the tumor has been transmitted directly through three generations. In the statistics reported here we have used only the cases appearing in the last generation. (*a*) The mother had her left eye removed during childhood for retinoblastoma, and the mother's father had his left eye removed for a tumor (confirmed histologically) during his childhood. Two children (a girl and a boy) both have bilateral retinoblastoma. A third child, born between the other two children, is healthy (Pedigree 2). (*b*) The father had his right eye removed at the age of 1 year and the left eye at the age of 2 years because of a retinoblastoma (confirmed histologically). The father's mother had her left eye removed at the age of 3 months, presumably because of a retinoblastoma. The one child, a girl, has bilateral retinoblastoma (Pedigree 3).

3. Our patient, not included in the series reported here, had bilateral retinoblastoma, but the parents have healthy eyes. Two maternal uncles, however, had unilateral retinoblastoma, confirmed histologically (Pedigree 4).

In conclusion, therefore, we can say that there is no contraindication to healthy parents who have had one child with retinoblastoma having more children, but there is a very strong contraindication to any retinoblastoma survivor having children.

‡ The clear circle indicates a normal eye; the black circle, an eye affected with retinoblastoma; the black circle with diagonal, an eye affected with retinoblastoma and enucleated.

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UNILATERAL BLINDNESS OCCURRING DURING ANESTHESIA FOR NEUROSURGICAL OPERATIONS

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SUDDEN unilateral blindness can appear during general anesthesia. In the eight cases reported in this paper the blindness followed neurosurgical procedures. These clinical cases and a series of experiments on monkeys corroborate the theories promulgated by previous authors on the etiology of this occurrence, that it is due to inadvertent application of pressure to the orbital contents.

All the patients in this series underwent operation at the Mayo Clinic during a 12-year period, from 1940 to February, 1952. No cases have occurred other than those reported, and none have appeared during other types of surgery.

Each patient had a lesion which required a suboccipital or posterior cervical approach. The patient either sat or lay prone with the face cushioned on a well-padded headrest. Intratracheal nitrous oxide and ether anesthesia was employed in every case. In spite of prolonged surgical procedures (two to six hours), the brachial blood pressure fell below normal in only two patients. There was minimal loss of blood. Each patient left the operating room in good condition.

On regaining consciousness, each patient complained of loss of vision in one eye. In most cases ophthalmologic consultation was obtained in less than 30 minutes. Each case in the series presented a remarkably similar clinical picture (Table).

At the initial examination, vision in the affected eye was nil in five patients and reduced to light perception in the remaining three patients. The opposite eye was invariably normal. Each patient had a dilated pupil which reacted consensually but not directly to light. The cornea was often slightly hazy. The eyelids usually were edematous. Five patients had proptosis of the involved eye. The edema and proptosis, when present, usually increased during the first 12 to 24 hours. Movements of the proptosed eye were always impaired, and in one patient the eye was completely immobile. The ipsilateral portion of the forehead was numb in two patients.

Ophthalmoscopic examination invariably revealed normal or dilated retinal arterioles, engorgement of the venules, and edema of the macula and of the retina surrounding the optic disc. The more severely involved eyes had a cherry-red spot in the fovea. The retinal edema increased during the first 12 to 24 hours and then

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Section of Ophthalmology (Dr. Hollenhorst) and Section of Neurologic Surgery (Dr. Svien), Mayo Clinic and Mayo Foundation; Fellow in Ophtha'mology, Mayo Foundation (Dr. Benoit). The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

was gradually absorbed in 7 to 10 days. The proptosis slowly receded, and the ocular rotations returned to normal during this period. After the first three or four days the arterioles began to narrow and become irregular; simultaneously, the optic disc showed the first signs of pallor. The optic disc of the permanently blind eyes became pearly white after 14 to 21 days. The arterioles narrowed to white threads. The macula and peripapillary retina became diffusely pigmented, and faint radial scarring appeared in the fovea.

Less severe cases were observed (Table). In these cases there was no proptosis, lid edema, or ocular paralysis. The immediate loss of vision was less severe. Mild macular edema was present. Partial or complete recovery of vision ensued.

Many hypotheses on the etiology of this condition were entertained. Because of the site of the surgical procedure, we at first considered the possible influence of excessive sympathetic stimulation. However, in most of the surgical procedures the surgeon had not been in the vicinity of the sympathetic trunks. Moreover, Unverricht [1] had previously stimulated the cervical sympathetic nerves in five of his

Unilateral Blindness *

Case	Eye	Anesthesia, Hr.	Blood Pressure	Initial Vision	Proptosis	Numbness of Forehead	Final Vision	Comment
1.....	L	2	N	L. P.	—	—	20/15	Full recovery
2.....	L	2	N	L. P.	—	—	20/20	Full recovery
3.....	L	2	N	Nil	—	—	20/60	Central scotoma
4.....	L	3	90/60	L. P.	++	—	H. M.	Preserved isles
5.....	L	3	N	Nil	++++	++++	H. M.	Preserved isles
6.....	L	4	N	Nil	?	+++	Nil	.....
7.....	R	4	N	Nil	++	—	Nil	.....
8.....	L	6	92/55	Nil	++++	—	Nil	.....

* N indicates normal; L. P., light perception, and H. M., hand movements.

patients. This produced ipsilateral dilatation of the pupil and widening of the palpebral fissure, but there was neither exophthalmos nor visual disturbance. Thrombosis or embolism of the ophthalmic artery was not a tenable hypothesis, since it could not account for the normal or dilated central retinal artery, the normal or dilated venous channels, and the evidences of active circulation through the retinal vascular tree. If vascular hypotension alone were to be implicated, the unilaterality of the phenomenon would be difficult to explain. Inadvertent pressure on the eyeball was considered but was difficult to prove, for none of the patients had corneal abrasions, bruises, or other signs of trauma. In fact, the heavily padded headrests indicated extreme care on the part of the anesthetist to protect the eyes.

The literature contains only three previous reports on the same subject. Slocum, O'Neal, and Allen [2] described a case in which they sectioned the fifth cranial nerve. During the operation, a portion of the Bailey headrest slipped over the right eye. To protect the globe, the anesthetist inserted a sponge between the eye and the headrest. The operation then continued for 1 hour and 15 minutes. When the patient awakened, this eye was blind. She later recovered the ability to see objects and detect color. These authors ascribed the loss of vision to accidental pressure on the eye in the presence of lowered blood pressure (80/60 mm. of mercury), due to gravity and loss of blood.

Givner and Jaffe [3] reported data on four patients in whom unilateral blindness followed abdominal operations with the patient under closed-mask anesthesia. The blind eye was always ipsilateral to the anesthetist and the anesthesia apparatus. Two of the four patients were in shock. Postoperatively, one patient had edema of the eyelid and another had a corneal abrasion. All had evidences of retinal ischemia. None recovered vision. These authors implicated pressure on the eye in the presence of shock and prolonged anesthesia. They attempted to reproduce the condition by pressure on the eye of rabbits and monkeys. Hemorrhages appeared in the retina of the rabbits, but no retinal infarction could be produced.

The two patients on whom data have been reported by Gillan [4] became blind in the left eye during closed-mask anesthesia. The systolic blood pressure of each patient was deliberately lowered to 50 mm. of mercury with hexamethonium for at least an hour to reduce surgical hemorrhage. One patient exhibited peripapillary and macular edema and had a cherry-red spot in the fovea. He did not recover vision. The other patient had mild macular edema but died four days postoperatively. Gillan ascribed the blindness to pressure on the eye in the presence of shock.

REPORT OF CASES *

CASE 1.—A 48-year-old man underwent operation in the prone position June 2, 1950, to remove an extradural protruded left seventh cervical intervertebral disk. Thirty minutes after cessation of anesthesia he awakened and complained of poor vision in the left eye. Immediate ophthalmologic consultation was obtained. The right eye was normal. The vision of the left eye was reduced to the perception of light. There was no lid edema or proptosis. The macula was faintly edematous, but otherwise the ocular fundus appeared normal. Five hours later the patient could detect hand movements. Twenty hours after cessation of anesthesia he could count fingers. Macular edema then covered an area 1 disc diameter in size and obliterated the foveal reflex, leaving a yellowish spot. The optic disc and retinal vessels were normal. Gross testing revealed a central scotoma measuring about 10 to 15 degrees.

By the third day the visual acuity of the left eye was 6/7. The fourth letter of four-letter words was omitted in reading. Tangent screen fields are shown (Fig. 1, left). On the 11th day the only abnormality of the visual field was mild enlargement of the blind spot (Fig. 1, right). The macular edema had been absorbed.

CASE 2.—A 29-year-old man underwent operation in the sitting position Sept. 2, 1950, for platybasia. On awakening, he complained of reduction of visual acuity to finger counting with his left eye. Confrontation fields disclosed a very small isle of preserved vision in the superior mid-periphery of the left eye. The retinal arterioles and veins had normal caliber. There was mild ischemic edema of the peripapillary and macular retina and a faint cherry-red spot in the fovea. Superotemporal to the optic disc was a small globular area of subretinal edema.

The following day the vision had improved to reading 14/141 print (A. M. A. reading card). The isle of vision had widened. Considerable edema was present in the macular area, but it had lessened near the optic disc. Forty-eight hours postoperatively vision had improved to reading 14/89 print; retinal edema had absorbed further. On the third day the patient read small print but omitted the first letter of each word. On the fourth day he read 14/21 print slowly, but by the sixth day with no difficulty. By the ninth day the visual acuity was 6/6 and the retinal edema had largely subsided. The visual field was plotted on the tangent screen on the 13th day; this showed enlargement of the blind spot and a temporal contraction (Fig. 2). The macula was clear, but slight edema persisted at the upper and lower poles of the optic disc.

A year later the visual acuity was 6/6. Ophthalmoscopic examination showed only minimal narrowing of the retinal arterioles.

* These cases are presented in increasing order of their severity, corresponding to the Table.

CASE 3.—A 65-year-old farmer underwent operation Jan. 12, 1951, in the prone position to remove a protruded sixth cervical intervertebral disk. Seven hours after anesthesia was terminated, the patient noted blindness of the left eye. There was no light perception. Very faint edema was observed between the optic disc and the macula. Oxygen inhalation, a stellate ganglion block, and intravenous administration of histamine were employed as therapy.

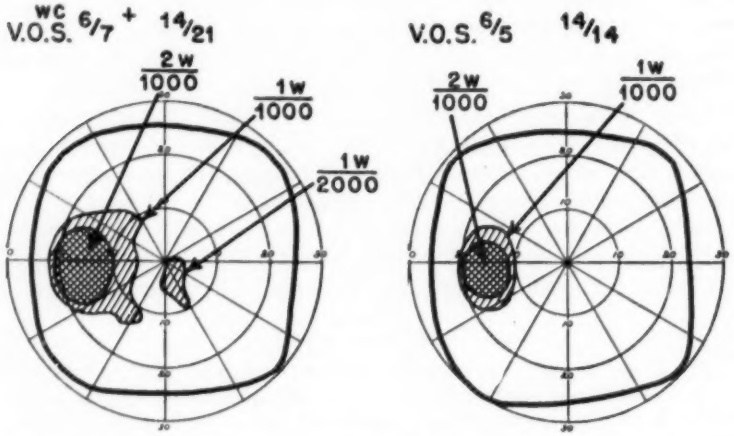

Fig. 1.—Visual field after anesthesia: (*a*) 3 days after operation; (*b*) 11 days after operation.

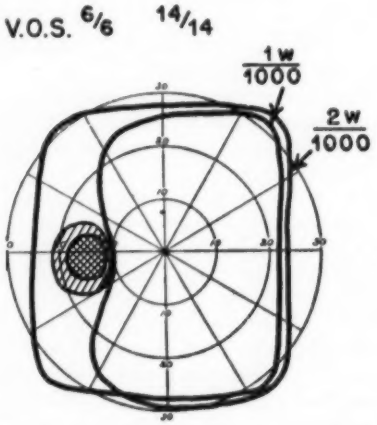

Fig. 2.—Visual field 13 days after anesthesia.

The next morning the patient could count fingers in the upper nasal field. There was ischemic edema between the disc and the macula and a faint cherry-red spot in the fovea. The optic disc and retinal vessels appeared to be normal. The ocular rotations were unimpaired.

By the third day the vision had improved slightly. An irregular focal constriction had appeared in the inferior temporal arteriole. By the fifth day the patient read large (14/224) test type. On the ninth day vision was 3/60. There was faint edema of the retina. The retinal arterioles of the left eye were very irregular. The disc had become pale.

On the 15th day the tangent screen field showed general depression and a large dense central scotoma (Fig. 3, left). The arterioles had narrowed further, and the focal constrictions had become very marked. The superior nasal arteriole was thread-like. The optic disc was moderately pale.

The final examination, on the 38th day, showed a residual central scotoma (Fig. 3, right). The arterioles were thread-like and very irregular. The optic disk was very pale.

Case 4.—A 50-year-old driver underwent operation Aug. 14, 1951, in the face-down position to relieve spinal pain resulting from metastatic carcinoma. A bilateral spinothalamic tractotomy was performed at the second thoracic level. The blood pressure remained at approximately 90/60 for three hours. Loss of blood was negligible.

On awakening two hours later the patient noted that he could perceive only light with the left eye. The eye protruded, and there was edema of the eyelids. The cornea was hazy. The eye was divergent, and rotations were impaired. There was milky edema of the retina and a cherry-red spot at the fovea. A stellate ganglion block was performed.

After 24 hours the cornea had cleared and the proptosis had receded. There was mild limitation of the ocular movements. The retinal arterioles had normal caliber. On the third day the patient could count fingers. On the sixth day the ocular rotations had returned to normal and

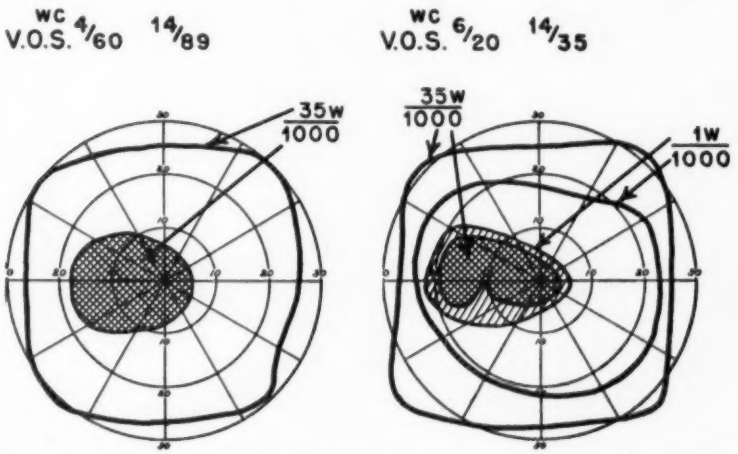

Fig. 3.—Visual field after anesthesia: (*a*) 15 days after operation; (*b*) 38 days after operation.

the edema of the lids had absorbed. The edema likewise was gone from the lower quadrants of the retina and had lessened elsewhere. The arterioles were irregular. By the eighth day there were severe focal constrictions in all the arterioles.

On the 16th day the patient could read very small (14/14) test type through a small isle of preserved vision centrally. The optic disc was moderately pale, and the arterioles were narrowed and irregular. The patient was dismissed and was not seen again.

Case 5.—A 39-year-old machinist underwent operation in the prone position Feb. 9, 1952, to relieve multiple protruded intervertebral disks in the first and second left thoracic spaces.

Four hours later the patient noted that the left eye was blind. The forehead on the left side was numb. There was edema of the eyelids. The eye showed 2 mm. of proptosis, and its movement was limited in all directions. In the macular region there was severe ischemic edema with a cherry-red spot. The arterioles had normal caliber.

On the following day there was more extensive retinal edema. By the third day the ocular rotations were improved and the macular edema had lessened. By the seventh day the arterioles had become moderately narrow and irregular. The retinal edema had been nearly absorbed. Light could be dimly perceived. On the 11th day the arterioles were extremely narrow and irregular; the veins appeared normal, and little edema persisted.

The patient was dismissed on the 17th day. At this time vision was limited to bare perception of hand movements in a tiny isle of the visual field nasal to the visual axis and in a lower temporal peripheral isle. The frontal anesthesia had not improved. There was mild limitation of ocular movement in the field of the inferior rectus muscle. No proptosis was present. There was moderate pallor of the optic disc, extremely narrow, irregular arterioles, but no residual edema of the retina.

CASE 6.—A 36-year-old woman suffered from severe Raynaud's disease. In 1940, a bilateral cervicothoracic ganglionectomy and sympathetic ramisectomy was done, with the patient in the face-down position.

Six hours later she complained of pain in the left eye and she could not raise the upper lid. The neurosurgical resident recorded redness of the eye and edema of the lids and postulated pressure from the face mask as the cause. The visual loss was not discovered until the following day. The left eye was blind. Proptosis was not recorded. The left side of the forehead was numb from the supraorbital margin to the vertex. There was widespread ischemic edema in the peripapillary and macular regions and a cherry-red spot at the fovea. The arterioles were slightly narrowed. The optic disc had normal color but blurred margins. By the fifth day the retinal edema had lessened and the arterioles had narrowed. On the seventh day the edema was almost gone, while the disc had begun to show pallor and the arterioles had become very narrow. At the time of dismissal, on the 14th day, no vision had returned to the affected eye. The optic disc was chalk-white; the arterioles and venules were thread-like; the fovea was scarred, and pigment disturbance was observed throughout the fundus.

CASE 7.—A 50-year-old foreman underwent operation Oct. 16, 1951, in the upright position in the Adson chair. A cervical laminectomy and decompression was carried out to relieve Klippel-Feil and Arnold-Chiari deformities, and a syrinx in the cervical portion of the cord was evacuated.

Three hours later the patient complained of blindness of the right eye. The eyelids were edematous. There was proptosis measuring 0.5 mm., and the ocular rotations were impaired nasally, temporally, and upward. Ophthalmoscopic examination revealed mild edema of the nasal portion of the optic disc, mild pale edema of the retina superotemporal and inferotemporal to the disc, engorged retinal veins, and slightly dilated arterioles.

Twelve hours later the proptosis had increased to 2 mm. The ocular rotations were limited, as before. The entire macular area had become edematous, and the retinal edema nasal to the disc had increased. The caliber of the arterioles was now normal.

By the fourth day the edema of the orbit was improved and the ocular movements were less limited. The arterioles were moderately narrowed. At time of dismissal, on the seventh day, there was further improvement in the ocular rotations, but 1.5 mm. of proptosis still remained. There was mild pallor of the optic disc and complete absorption of the edema from the peripapillary retina, but still some edema in the macula. The arterioles were very narrow and irregular.

A letter from the patient four months later stated that not even light perception had returned to the right eye.

CASE 8.—A 29-year-old painter underwent operation in January, 1941, for relief of hyperhidrosis. In the prone position, bilateral thoracic sympathectomy and ramisectomy of the first, second, and third thoracic ganglia were carried out.

Three hours later the patient complained of visual loss in the left eye. There was complete internal and external ophthalmoplegia, with proptosis of the blind eye. As in the previous cases, there was a milky-white, edematous retina with a cherry-red spot at the macula. The arterioles and veins had normal caliber.

The following morning there was chemosis of the conjunctiva, edema of the eyelids, and further exophthalmos. On the third day slight motion of the left eye was possible. On the fifth day the proptosis was unchanged but there was less chemosis, less retinal edema, and some pigment migration into the macula. By the eighth day there had been no further improvement in the ocular rotations but the eye appeared normal externally. The retinal edema had improved, and the arterioles had narrowed. There was much pigment mottling deep in the retina and choroid.

On the 12th day all the chemosis of the conjunctiva and edema of the eyelids had disappeared. The ocular rotations had improved. The optic disc was moderately pale. The retinal arterioles were thread-like, and some were obliterated. Slight edema of the retina remained, and there was pigment stippling all over the fundus.

On the 19th day there was still slight residual edema at the upper and lower poles of the optic disc. The disc had a chalky-white appearance. There was no recovery of light perception.

The very strong evidence that unilateral blindness in these eight patients resulted from pressure transmitted to the eye impelled one of us (Svien) to modify the design of the face rest. The essential changes were the widening of the crossbar on which the forehead rests, the separation of the two lateral bars, and the removal of the padding over the orbits so that the eyes would not be touched by the face rest (Fig. 4*a* and *b*). In the two and a half years since this modification was made not a single additional case of this type has occurred.

Fig. 4.—(*a*) Face rest used for surgical procedures reported; (*b*) face rest as modified.

EXPERIMENTAL PROCEDURES AND RESULTS

The reproduction of this sequence of events in experimental animals was carried out by two of us (Hollenhorst and Benoit). A detailed account is given by Benoit.† The monkey (Macacus rhesus) was employed. We had great difficulty at first in our attempts to reproduce this condition. After the correct technique was developed, the entire syndrome could be produced at will in the majority of attempts. Photographs of the monkey fundus were made with the Zeiss-Nordenson retinal camera as modified by Ogle and Rucker.[5] The essential improvement in the camera is the replacement of the original filament incandescent lamp by a high-speed flash tube. Reflex-free pictures are obtained. Since the duration of exposure is about 0.0001 second, the problem of movement of the eye is eliminated.

† Benoit, C. F.: Retinal Changes Associated with Pressure on the Eyeball During Anaesthesia, Thesis, Graduate School, University of Minnesota, 1954.

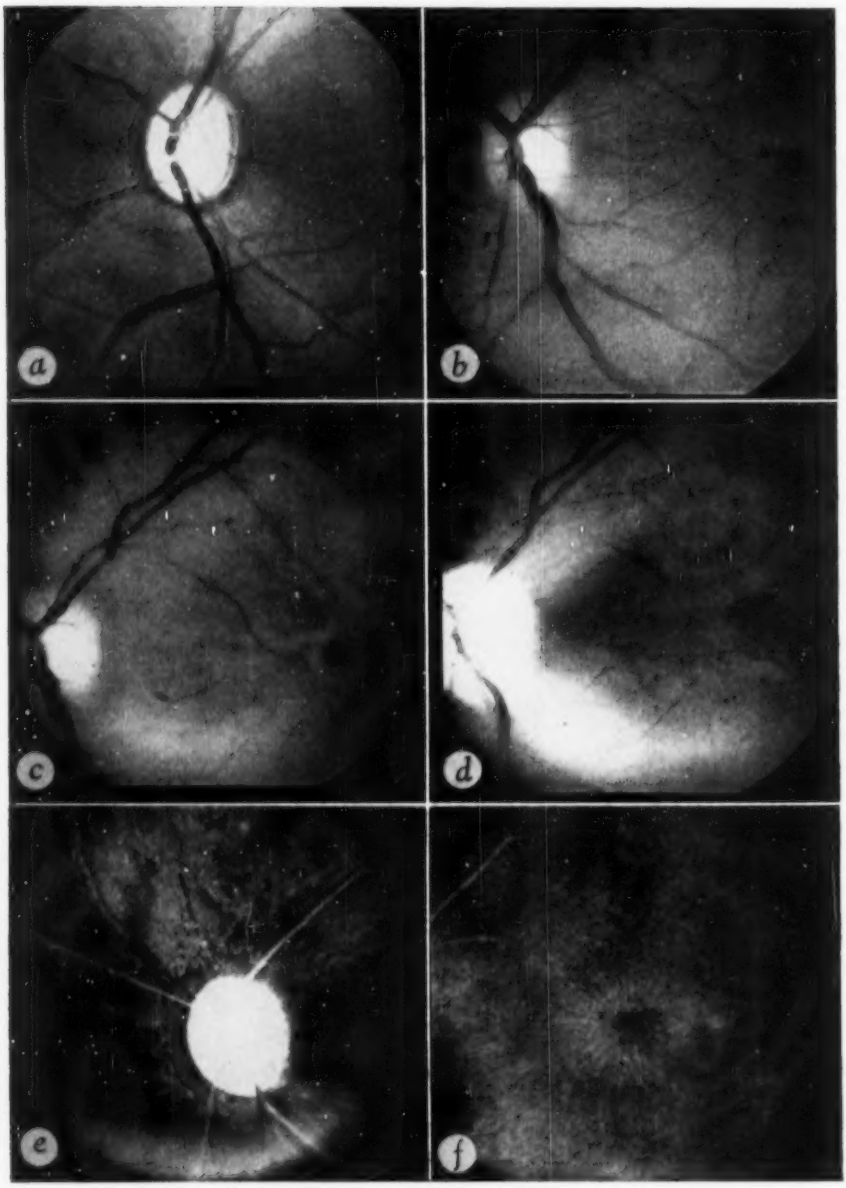

Fig. 5.—Fundus of a monkey. (*a*) Normal fundus; (*b*) fundus 6½ hours after infarction; (*c*) fundus 48 hours after infarction, (*d*) fundus 9 days after infarction; (*e*) optic disc 120 days after infarction; (*f*) macula 120 days after infarction.

The typical procedure and course are described in one experimental animal. Figure 5*a* shows a normal fundus of a monkey.

A 4.3 kg. monkey was anesthetized with intravenous pentobarbital sodium. The femoral artery was cannulated and connected with a mercury manometer. Then 43 cc. of blood was removed from the artery. This caused the blood pressure in the femoral artery to fall from 140 to 126 mm. of mercury. Then we placed a rubber bag connected to a manometer between two layers of sponge rubber and tightly applied this apparatus to the orbital contents with a bandage. The rubber bag was then inflated to exert a pressure of 200 mm. of mercury on the orbit. Then 5 mg. of hexamethonium chloride injected subcutaneously lowered the blood pressure in eight minutes to 66 mm. of mercury. The orbital compression was maintained 60 minutes. The blood pressure varied between 66 and 88 mm. of mercury. At the end of the experiment 43 cc. of blood was transfused back into the animal.

Because of marked ocular hypotony, the retinal structures could not be visualized immediately after the pressure was released. About two hours was necessary for clearing of the media and reestablishment of the intraocular pressure. By this time there was edema of the eyelids and 2 mm. of proptosis of the left eye. Ophthalmoscopic examination revea'ed massive pale, ischemic edema involving the peripapillary and macular areas. In the fovea was a cherry-red spot. The retinal arterioles and venules were dilated to twice their normal size.

During the following three and a half hours (six and a half hours after the initial application of the pressure) there was an increase in the edema of the retina which almost obliterated the cherry-red spot (Fig. 5*b*). The eye was still slightly hypotonic. The orbital tissues had become so edematous that the eyeball was fixed in the orbit. The proptosis then measured 4 mm.

Twenty-four hours later the monkey was active and alert. Intraocular tension was normal. The retinal edema had become even more pronounced. Several punctate hemorrhages had appeared near the optic disc. The arterioles were one and a half times the normal size.

Forty-eight hours after the experiment the proptosis was less severe and 50% of ocular movement had returned. The retinal arterioles were narrowed Grade 1 (Fig. 5*c*).

Seventy-two hours after the experiment the ocular rotations were normal. The retinal edema had begun to subside.

On the fifth day the proptosis was no longer present. There had been further absorption of the retinal edema.

On the seventh day there was mild pallor of the optic disc. The retinal arterioles were narrowed, Grade 2.

On the ninth day there was moderate pallor of the optic disc. We could then observe edema in the retina only along the superior and inferior temporal vessels. There was beginning migration of pigment into the macula. The retinal arterioles were narrowed, Grade 2 (Fig. 5*d*).

On the 14th day there was severer pallor of the optic disc and the arterioles were even narrower (Grade 3). There was very slight retinal edema.

On the 21st day the retinal arterioles showed narrowing of about Grade 3. The optic disc was very pale. The macula was scarred.

From the 50th to the 120th day the retinal arterioles became thread-like and sheathed. The optic disc was chalky-white (Fig. 5*e*). There was diffuse pigmentation of the retina. The macula was scarred (Fig. 5*f*).

Although this syndrome could be produced almost at will with the procedure described, there were occasional failures. This occurrence could not be explained satisfactorily. We could not determine with certainty the essential conditions for producing this type of retinal ischemia. In our experiments three conditions seemed necessary: (1) correct application of pressure to the orbital contents, (2) removal of a quantity of the circulating blood, and (3) lowering the blood pressure with hexamethonium chloride.

We produced retinal ischemia in one monkey which may not have met these three conditions. Pressure was applied to the left eye of this monkey for 60 minutes, and at the same time blood was removed and hexamethonium injected. However, no retinal ischemia resulted. About three hours later we reanesthetized the same monkey and applied pressure to the right eye for 60 minutes but removed no blood and injected no hexamethonium. The typical syndrome resulted. Unfortunately, the status of the blood pressure was unknown, since we have not inserted a cannula into the femoral artery.

The minimal duration of pressure on the orbit necessary to produce retinal ischemia was not determined. In this series, the shortest effective time of application was 60 minutes. Theoretically, retinal ischemia for as brief a time as 7 to 10 minutes should produce visual loss. Duane,[6] observing the fundus oculi during visual blackout on the human centrifuge, demonstrated that the visual function of the retina ceases two to five seconds after the retinal blood supply is impaired.

The eye of one monkey was enucleated 24 hours after retinal ischemia was produced. Another specimen was removed 120 days afterward. Representative sections are shown to illustrate the extremely marked retinal edema in the early stages (Fig. 6*a* and *b*), in contrast with the severe retinal and optic nerve atrophy which takes place later in the course (Fig. 6*c* and *d*). It is interesting to note the widely dilated vascular channels in the early stages.

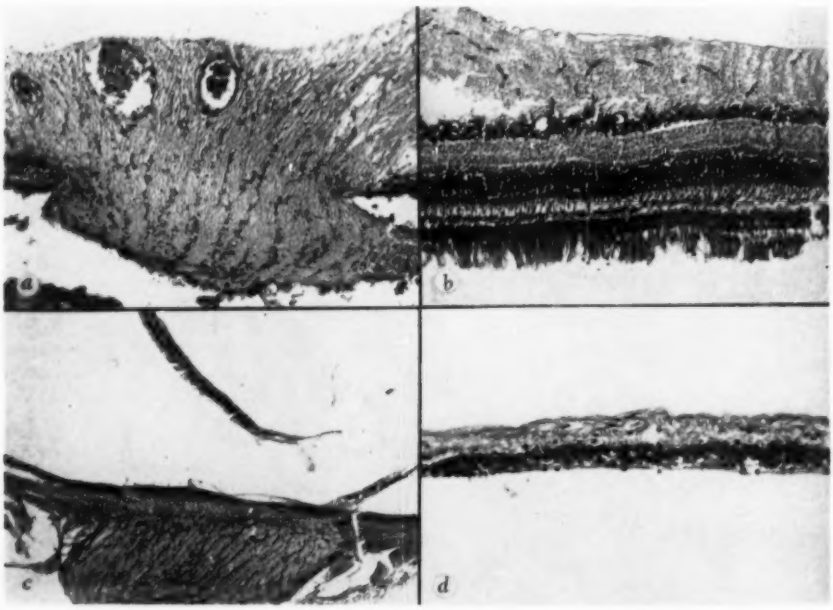

Fig. 6.—Sections from eyes of monkeys after infarction. (*a*) Optic disc 24 hours after infarction (× 62); (*b*) retina 24 hours after infarction (× 80); (*c*) optic disc 120 days after infarction (× 56); (*d*) retina 120 days after infarction (× 140).

These experimentally produced ocular lesions, which so closely simulate the events observed in the eight clinical cases, appear to be almost incontrovertible evidence that the mechanism is identical in man and in monkey. The common denominator is the application of pressure to the eyeball. Whether or not a state of shock is a necessary condition has yet to be proved, although in monkeys both excessive loss of blood and deliberate lowering of the blood pressure with hexamethonium seem to be necessary. Blood pressure levels consistent with a state of shock were demonstrable in only two of the four cases reported by Givner and Jaffe, and in the present series the blood pressure levels remained normal in six of the eight patients.

It is, of course, possible that even though the recorded brachial blood pressures were normal, there may have been a markedly lowered pressure in the internal and external carotid artery systems, so that a state of "shock" was actually present in the orbital circulation.

The disturbances of physiology accompanying these events seemingly are initiated by a partial or complete collapse of the arterial and venous channels in the orbit produced by a tamponade action of the ocular contents. When the external pressure is released, the ischemic vascular channels dilate and there is a transudation of fluid through the permeable walls into the tissue spaces. This results in orbital edema, proptosis, paresis of ocular movement, and massive edema of the retina. Because of the extremely low tolerance of the ganglion cells to ischemia, loss of vision probably precedes all the other phenomena and occurs in the absence of orbital signs. If the retinal ischemia were of only short duration, some recovery of vision would be anticipated, as noted in Cases 1 through 5. The relative anoxia of the arteriolar and venous walls probably accounts for their invariable state of dilatation when seen in the early stages of this disorder.

In the following weeks there is gradual absorption of edema. Progressive sclerosis and narrowing of the previously ischemic retinal arterioles lead finally to sheathed, thread-like vessels. The ganglion cells of the retina degenerate, and there is total atrophy of the optic nerve in the severer cases.

Positioning of the patient's head probably accounts for the preponderance of left-sided involvement. The surgeon usually stands on the left side of the patient, and this may contribute to compression of the left orbit.

At the Mayo Clinic, intratracheal anesthesia is preferred for long surgical procedures of all types. Closed-mask anesthesia is rarely employed. Posterior cervical and suboccipital operations are the only procedures in which a face rest is used. These facts probably explain the absence of cases of unilateral blindness during anesthesia in any other type of operation.

SUMMARY AND CONCLUSIONS

Sudden unilateral blindness occurred in eight patients during anesthesia. Each patient underwent a suboccipital or posterior cervical operation under general intratracheal anesthesia. Each patient sat up or lay prone with the face in a face rest. After awakening from the anesthesia, each patient complained of unilateral loss of vision. This was found to be due to retinal ischemia. Varying degrees of lid and orbital edema with proptosis occurred. The final visual status of the affected eye ranged from complete recovery of vision to permanent blindness.

Inadvertent pressure on the ocular structures, and possibly lowered blood pressure in the orbital arteries, is the cause of this condition.

We reproduced in the monkey the identical sequence of events by lowering the systemic blood pressure with subcutaneous hexamethonium chloride and by bleeding the animal while simultaneously exerting pressure on one orbit.

Fundus photographs illustrate the intraocular changes observed in the monkey. These are identical with the clinical findings in human beings. Photomicrographs illustrate the changes in the retina which occur after 24 hours and after 120 days.

A modification in the design of the headrest has helped prevent any additional human cases for two and a half years.

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DIFFERENTIAL RADIOPHOSPHORUS UPTAKE OF LENS

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THE PRESENCE of lens epithelium on the anterior lenticular surface only has led to speculations concerning the possibility of posteroanterior nutrient flow through the lens. Besides the obvious physiologic importance of such a nutrient flow, there exist many pathologic implications—for example, it would be attractive to consider cataract as a sequel to metabolic or senile changes of the lens epithelium. In an attempt to demonstrate whether such a posteroanterior metabolic gradient exists, the concentration of radioactive phosphorus in different portions of the rabbit lens was studied by the following techniques.

METHODS

Adult albino rabbits were given radioactive phosphorus (P^{32}) intravenously in the form of acid phosphate. After the given time (see accompanying Table for dosage of P^{32} and time allowed for absorption into lens) the eyes were enucleated, with local use of procaine anesthesia. Immediately after enucleation the lenses were removed without injury to the capsule, rinsed briefly in water to remove "hot" aqueous contamination, quick-frozen with solid carbon dioxide, and sectioned equatorially into anterior and posterior halves. In the first four eyes the equatorial portion of each half was pared off and discarded, since the comparison desired was the radioactivities of the anterior and the posterior lens and the equatorial zone was considered as possibly having in-between radioactivity, reducing the likelihood of demonstrating a significant difference. The epithelium and capsule were left on the first four eyes. From the last four eyes the capsule and epithelium were carefully removed, and the anterior capsule and epithelium were saved and measured.

The specimens (anterior lens, posterior lens, and, for the last four eyes, anterior capsule and epithelium) were weighed, in the fresh state, to the accuracy of 0.1 mg. Each individual specimen was mechanically macerated, then digested for 12 hours in 2 cc. of fuming nitric acid with one drop of 30% hydrogen peroxide (Superoxol) added. Digestion was carried on in a boiling-water incubator, evaporation being prevented by a "cold finger" (a tube of running cold water in the mouth of the digesting tube to condense and return all evaporation from the sample). Specimens were then neutralized with 0.1 N sodium hydroxide and diluted to a standard total volume of 5 cc. A 1-cc. aliquot was placed in each of three weighed planchets, evaporated, with avoidance of splatter loss, and counted to a total of 6,400 with a thin end-window Geiger-Müller tube. Planchets were reweighed and corrected for background, self-absorption, and decay. All values were corrected to counts per minute per 100 mg. of tissue.

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RESULTS

The accompanying Table presents the values obtained. It is noted that counts were low, and often unobtainable, in the first three eyes, which received a low dose of P^{32} for short times. The anterior half of Lens 4 showed double the radioactivity of the posterior half. This was believed to be due to the epithelium, which was removed and measured separately for the last four eyes. The epithelial radioactivity was found to be enormous—in fact, 100 times that of the remainder of the lens for Lenses 5 and 6. The lower values for Lenses 7 and 8 are due to faulty

Values Obtained in Differential Radiophosphorus Uptake in Lens

Eye		P^{32}, μc	Time Between P^{32} Injection and Enucleation, Min.	Wet Weight, Mg.	Counts/ Min./Mg.	Average Counts/ Min.
1	Anterior	50	6	32.5	.....	...
	Posterior	..	..	35.8	.....	...
2	Anterior	50	14	60.8	18.09	...
	Posterior	..	..	48.4	.....	...
3	Anterior	50	30	41.2	12.13	...
	Posterior	..	..	47.6	16.8	...
4	Anterior	50	38	44.0	245.45 216.78	231
	Posterior	..	..	45.4	123.34 110.13	111
5	Anterior	100	30	122.7	553.8 563.2	558
	Posterior	..	..	159.5	418.4 402.8	410
	Epithelium	..	..	1.6	42,083.75 41,809.50	41,946
6	Anterior	100	40	175.1	375.23 366.96	371
	Posterior	..	..	143.4	471.75 498.05	484
	Epithelium	..	..	1.3	57,049.2 60,223.0	58,636
7	Anterior	100	45	159.2	479.81 502.62	491
	Posterior	..	..	151.4	507.52 493.71	500
	Epithelium	..	..	6.4	10,630.3 10,627.1	10,628
8	Anterior	100	55	111.5	637.83 610.56	624
	Posterior	..	..	139.6	574.65 517.89	546
	Epithelium	..	..	12.0	6,394.5 6,687.6	6,541

removal of the epithelium, which was obviously contaminated by cortex, as evidenced by the excessively large wet weight of the epithelium for Lenses 7 and 8. No significant difference was demonstrable in the concentrations of P^{32} in the anterior and posterior portions of the lenses. This experiment cannot, therefore, be used to support a theory of posteroanterior nutrient flow in the lens.

SUMMARY

With the aid of radioactive phosphorus, rabbit lenses were examined for evidence of a posteroanterior nutrient flow. Under the conditions of this experiment, such a posteroanterior gradient could not be demonstrated to exist for phosphorus. The lens epithelium was found to absorb radioactive phosphorus to a concentration 100 times that of the rest of the lens.

ETIOLOGY OF RETROLENTAL FIBROPLASIA

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ROTTERDAM

AN ANATOMICAL investigation on eyes of premature infants, with the purpose of finding the anatomical features of the earliest stage of retrolental fibroplasia, was started in 1951. A total of 114 eyes of 59 babies were examined. None of the infants had been examined ophthalmologically during life. The birth weights and the ages of the babies were as follows:

Birth Weight, Gm.	No.	Age	No.
500-1,000	15	0-10 days	47
1,000-1,500	31	10-20 days	3
1,500-2,000	12	20-30 days	1
2,000-2,500	1	1-2 mo.	4
		2-3 mo.	3
		3-4 mo.	1

The earliest pathologic stages of the disease had already been described by Heath [1]; Tyner and Frayer [2]; Reese and Blodi [3]; Reese, Blodi, and Locke,[4] and Friedenwald, Owens, and Owens.[5] We found typical lesions of the first stage of the disease in six eyes of three children. The finding of three cases of retrolental fibroplasia in 59 unselected autopsies on premature infants confirms the opinion of von Winning [6] that the incidence of the disease in the Netherlands is not below that in the United States. It may be significant that the three children all were about 2 or 2½ months of age.

Figure 1 *A* to *E* represents in chronological order the earliest changes in the retina. The very first changes are characterized by retinal edema and proliferation of the capillary-forming mesenchymal cells in the nerve fiber layer. This proliferation may be local, with a whorl-like arrangement, or may be more diffuse. The capillary buds and the whorl-like proliferations break through the internal limiting membrane and grow along the inner side of the retina, forming a preretinal capillary plexus. Case 1 did not show retinal hemorrhages, and only minimal signs of gliosis were found in the retinas in Cases 1 and 2. Retinal hemorrhages and gliosis seem, therefore, not to be significant in the first stage of the disease. The capillary proliferations in these two cases were not limited to the zone of the oral serrata, but were present also in the posterior eye segment. The eyes in Case 3 showed a slightly more advanced stage, with a heavily vascularized membrane arising from the retina in the region behind the ora serrata. No anomalies were found in the posterior eye segments in this case.

This study was supported by a grant from the Netherlands Health Organisation T. N. O.

Ryan [7] thought delayed retinal coaptation to be an important etiologic factor in retrolental fibroplasia. We found, however, folds of the inner retinal layers in 102 of the 114 eyes examined. These retinal folds seem to be postmortal features, and not characteristic of retrolental fibroplasia.

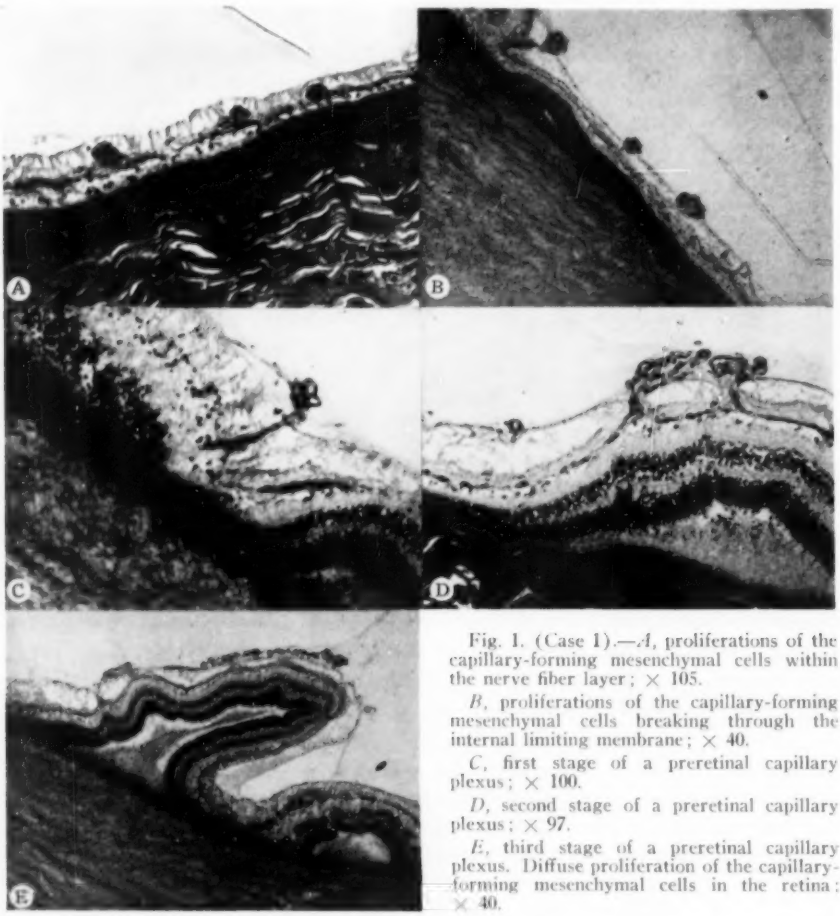

Fig. 1. (Case 1).—*A*, proliferations of the capillary-forming mesenchymal cells within the nerve fiber layer; × 105.

B, proliferations of the capillary-forming mesenchymal cells breaking through the internal limiting membrane; × 40.

C, first stage of a preretinal capillary plexus; × 100.

D, second stage of a preretinal capillary plexus; × 97.

E, third stage of a preretinal capillary plexus. Diffuse proliferation of the capillary-forming mesenchymal cells in the retina; × 40.

PATHOGENESIS OF RETROLENTAL FIBROPLASIA

In 1952 Friedenwald stated that he preferred not to discuss the pathogenesis of the disease and what it was that caused the blood vessels to proliferate in the direction they did, as it seemed to him that enough was not known to permit more than speculation in that field. Now, however, more is known about many aspects of these problems, and it seems possible to discuss both questions without being speculative.

Since Szewczyk * and Ryan,[7] at the same time, in 1952, focused attention on the role of oxygen in the etiology of retrolental fibroplasia, many papers have appeared which suggest that a defect in the oxygen metabolism of the retina in the premature infant plays a part in the development of the disease. Szewczyk thought that retrolental fibroplasia is a response of immature neural tissue to anoxia. Ryan, on the contrary, thought that the disease was due to the administration of excess oxygen. Since then, some have held the disease to be due to anoxia, relative anoxia, or hyperoxia, while others believe that oxygen plays no part in production of the disease.

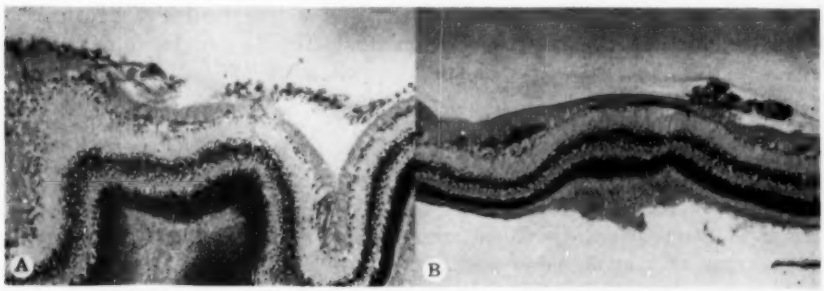

Fig. 2 (Case 2).—*A*, origin of the preretinal capillaries from the capillaries in the nerve fiber layer; × 60.

B, photograph from the other eye; × 60.

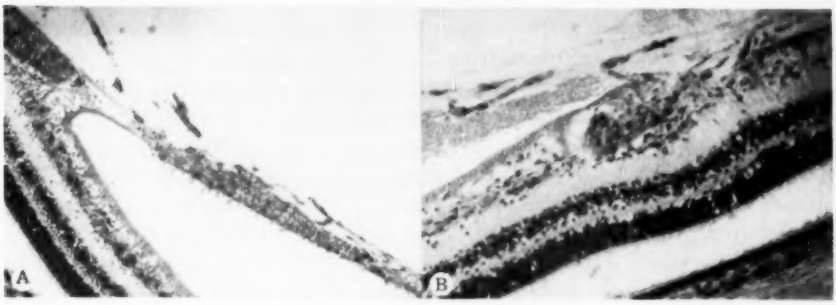

Fig. 3 (Case 3).—*A*, heavily vascularized membrane arising from the retina in the region behind the ora serrata; × 60.

B, photograph from the other eye. The origin of the preretinal capillaries from intraretinal proliferations is clearly visible; × 125.

Evidence that the administration of oxygen influences the onset and the course of the disease is extremely suggestive. It has appeared from many reports that retrolental fibroplasia is related to the administration of oxygen or its sudden withdrawal. These reports are discussed in the exhausive reviews by Zacharias,[10] Maumenee,† and Wagener.[13] It also has been shown in recent years that the disease can be largely prevented by curtailing the amount of oxygen given.

* References 8 and 9.

† References 11 and 12.

A close correlation between the use of oxygen and the onset of the disease exists, however, only in a minority of cases. This has been demonstrated by the findings of Silverman, Blodi, Locke, Day, and Reese.[14] These authors discovered the first symptoms in 11 infants while they were under continuous oxygen administration, in 15 infants during the first week thereafter, and in 17 infants during the second week, in 13 infants during the third week, and in 13 infants more than three weeks, after the cessation of oxygen administration.

A fruitful task is reserved for pediatricians to determine the partial pressure of oxygen in the arterial blood and the oxygen saturation of hemoglobin in children in whom retrolental fibroplasia is developing. It is, however, possible to approach the problem from other directions, and it appears that the anatomical, as well as the physiological and the clinical, side of the problem only provides arguments for the view that anoxia or hypoxia is the ultimate stimulus to the proliferation of the capillary-forming mesenchymal cells in the nerve fiber layer of the retina and to the growth of newly formed blood vessels into the vitreous.

ANATOMY

As stated, all anatomical investigations have shown that the first stage of the disease is characterized by retinal edema and proliferation of capillary-forming mesenchymal cells in the nerve fiber layer.

Michaelson [15] has demonstrated the following features in the normal capillary development in the retina:

1. The formation of retinal capillaries is preeminently a function of the retinal veins.

2. If vein and artery are close to each other, growth takes place initially from the side of the vein more remote from the neighboring artery.

3. The extension of growth toward an artery is limited, leaving, finally, in the definitive eye a well-marked capillary-free space around the arteries.

These features of the capillary development in the retina have led to the postulate that the higher oxygen tension which might exist around a retinal artery inhibits further extension of capillaries during development and so accounts for the clearly demarcated capillary-free zone. Campbell [16] has tested this hypothesis on the rat, in which the retinal vessels are just commencing to develop from the hyaloid vessels at birth. Twenty young virgin female rats were mated with two young male rats. Each alternate litter born was placed in a low-pressure chamber 24 to 30 hours after birth. The remaining litters were used as controls. The partial oxygen tension in the air of the chamber was 76 mm. Hg. The width of the capillary-free zone in control and in experimental animals of comparable age was measured: In the control group 134 measurements resulted in a mean width of 63.19 μ; in the low-pressure group 116 measurements gave a mean width of 49.12 μ. This difference is highly significant.

The difference in the capillary-free zones can be explained by postulating that the oxygen tension in the tissues around the developing vascular bed controls to some extent the mode of formation of the developing vessels. When the developing capillaries reach an area of retina with a relatively high oxygen tension, further extension ceases. In the low-pressure group the capillaries could grow nearer the arteries, presumably because the lower oxygen tension in the arterial blood stream

causes a decrease in the diameter of the surrounding zone which is adequately supplied with oxygen. From the investigations by Michaelson and Campbell the conclusion may be drawn that the retinal blood vessels grow toward an area of low oxygen tension and cease to extend when an area of higher oxygen tension is reached.

Ashton, Ward, and Serpell,[17] in experiments on kittens, have shown that high concentrations of oxygen obliterate the retinal vessels. The extent of the effect of oxygen was directly proportional to the degree of immaturity of the retinal vascularization, to the duration of the exposure to oxygen, and to the degree of oxygen concentrations. The obliterated retinal vessels attempted to reopen when the kittens were replaced in air, but the obliteration appeared to be irreversible in many vessels. Thus, retinal ischemia was induced, and an abnormally excessive growth of vessels advanced wildly from the disc and extended into the vitreous. According to Ashton,[18] the present evidence is in favor of anoxia or hypoxia as the stimulus to vascularization.

This evidence is confirmed by the work of Patz,[19] who produced the characteristic lesions of human retrolental fibroplasia by exposing various sorts of young animals to an increased oxygen environment. The mechanism of oxygen action on the retina is summarized as vasoconstriction with production of decreased retinal blood flow. The ultimate stimulus to endothelial proliferation in these experiments has been retinal hypoxia, produced by an excess of environmental oxygen.

All anatomical knowledge on the development and proliferation of retinal capillaries is therefore in favor of the view that the first stage of retrolental fibroplasia is due to retinal hypoxia.

Nothing is known about the oxygen tension in the vitreous and in the choroid, but it may be assumed that the oxygen tension in the avascular vitreous is lower than in the highly vascularized choroid. The answer to the question why the newly formed capillaries in retrolental fibroplasia grow in the direction of the vitreous may therefore be that the capillaries grow in the direction of the lowest oxygen tension.

Campbell in part started from the same assumption when he noted that the retinal capillaries penetrate only as far as the inner nuclear layer in the human retina, stating, "Could the cessation of growth in this plane also be influenced by the gradient of oxygen tension arising from the choroid?"

PHYSIOLOGY

The superficial and irregular breathing of premature babies makes it probable that for some periods a large proportion of the tidal air never ventilates the alveoli. Malm [20] thinks that the unsatisfactory alveolar ventilation is caused to an important extent by the disadvantageous ratio between the volume of the respiratory passages ("dead space") and that of the alveoli. He found that the ratio of dead space to the respiratory capacity of a premature infant weighing 1,690 gm. is 1 : 2.5, whereas the corresponding ratio for a full-term baby weighing 3,500 gm. is 1 : 13.6. The deleterious effect of even a small reduction of the respiratory surface, as, for example, in pneumonia, becomes quite evident against this background.

All premature infants are born with "canalar" lungs, in which the air crevices correspond to the alveolar passages of the adult lung. These air crevices differ from true alveoli in the existence of a border of cuboidal epithelium, more or less continuous, according to the fetal age at birth (Lelong and Laumonier [21]). The intercanalar mesenchymatous septa are very thick and contain numerous cells and several capillary networks, whose contact with air is inversely proportional to the degree of prematurity. The premature infant develops its true alveolar system only after the date of its full term; maturation does not appear to be hastened appreciably by respiration in air; the process of maturation requires the same time as it does in the fetus. Respiratory function is possible in the canalar lung, but the structure indicates that this function operates under difficult conditions. There are, in fact, two obstacles to exchanges between air and blood: the epithelial canalar border and the complex route of the rich capillary network situated in the mesenchymatous mass, with a partial contact between the vascular loops and the gaseous cavities.

Darling, Smith, and Young [22] found the oxygen-carrying capacity of the blood of two premature infants of 5 and 7 months, respectively, lower than the mean value in a group of mature infants. Jonxis [23] found the oxygen saturation of the blood in premature and mature infants on the same level as that of adults, viz., 97%. However, in young premature infants the oxygen saturation of the arterial blood oscillates between still wider limits than in adults. An abrupt fall in the oxygen saturation of about 4% is occasionally seen.

In adults the oxygen tension in the alveolar air is about 100 mm. Hg. The partial oxygen tension of the air is 20% of 760 mm. Hg, or 152 mm. Hg. Thus, one-third of the oxygen of the inhalated air in the adult has already been lost before the air comes in contact with the pulmonary capillaries. The oxygen tension in the alveolar air of premature infants is not known, but this oxygen loss will probably be the same.

All these facts make it clear that the respiratory function of the premature lung operates under very difficult conditions. It seems, therefore, difficult for the premature baby to get an abnormally high concentration of oxygen in the arterial blood, even in environmental high oxygen concentrations. Jonxis does not believe that variations of between 40 and 60% oxygen concentration would have much effect on the infant's arterial oxygen saturation. Brinkman [24] thinks it very difficult to produce toxic tensions of oxygen in the body at normal pressures, and it appeared that no delegate on the symposium on "Anoxia of the Newborn Infant" [25] had ever seen clear indications of oxygen poisoning in infants, although considerable hyperventilation was observed in infants in atmospheres of 60 to 100% oxygen.

CLINIC

In healthy premature infants the retinal vessels are attenuated and straightened when the child is placed in high oxygen concentrations, and the vessels show dilatation and tortuosity when the child is placed in low oxygen concentrations. The clinical course of retrolental fibroplasia has been described in many reports as beginning with dilatation and tortuosity of the retinal vessels.

A dramatic improvement has often been noted when babies with ophthalmoscopic changes of short duration were treated with high oxygen concentrations. Such improvements have been described by Szewczyk, Swart-van der Hoeven and

Mak [26]; Jefferson [27]; Huggert,[28] and Bedrossian, Carmichael, and Ritter.[29] Recurrences have been described when the babies were replaced in lower oxygen concentrations, and improvement again occurred when high oxygen concentrations were readministered. Bedrossian and associates even described an infant to whom oxygen therapy was given three times, with successful results, because of recurrence on each withdrawal. These clinical facts, also, are an impressive plea for the concept that retrolental fibroplasia is due to a retinal hypoxia.

The literature shows a remarkable lack of detailed clinical histories. When they are given or when they are reviewed, it appears that the babies in a large majority of the cases suffered from severe anemia or pneumonia. Szewczyk has emphasized the important role of anemia and respiratory infections, or of conditions interfering with respiration, in the occurrence of retrolental fibroplasia. Statistics presented by Apple [30] have shown that 5 out of 9 premature babies who developed retrolental fibroplasia had a hemoglobin percentage of below 50, as against 3 out of 17 premature infants who did not develop the disease. Bedrossian and associates found that respiratory infections were directly associated with the course of the disease in 12 infants.

CLINICAL REPORT OF CASES

The clinical histories in our cases illustrate that it is necessary to consider the whole infant, and not its eyes or its incubator only, in order to obtain an insight into the etiology of retrolental fibroplasia.

CASE 1.—G. v. S., a girl, was born spontaneously on April 18, 1952. The birth weight was 1,290 gm. The child was immediately removed to an incubator (Isolette). By April 26 the weight had decreased to 1,025 gm.; it increased slowly afterward. The general condition remained satisfactory during the following weeks, despite somewhat too frequent stools. On May 12 the child became seriously ill, and it seemed that she would die. She was cyanotic, and respiration had become very superficial. Oxygen administration was started, with a flow of 8 liters a minute. She recovered within half an hour. From May 16 to May 18 the oxygen flow was reduced to 6 liters a minute, and afterward to 4 liters a minute. On May 27 the weight had increased only to 1,160 gm.

A small abscess had developed on the back after a subcutaneous injection. On May 19 the abscess had to be incised, and again on May 29, June 6, and June 10, despite penicillin therapy. The hemoglobin content had fallen to 29%, and on June 4 a blood transfusion, of 18 cc., was given. Minor edema developed on the back of the hands and feet, and the child died on June 11.

CASE 2.—M. J., a girl, was born on Feb. 1, 1952, by Caesarean section, necessitated by maternal eclampsia. The weight was 1,390 gm. at birth; it decreased to 1,200 gm. by Feb. 6, and the birth weight was regained by Feb. 18. On Feb. 26, for the first time, the child became cyanotic; she recovered quickly after removal to a Gordon-Armstrong incubator, where she received oxygen. The quantities of oxygen given were not noted, but it appears from the clinical history that the child did not do well without oxygen on March 15. About this time fine rales were heard over the left lung. On March 20 the hemoglobin content had fallen to 36%. The same day a blood transfusion of 36 cc. was given, and again on March 22. On March 27 the hemoglobin content was again reduced, to 50%. The condition became worse, and serious dyspnea developed. The child was treated with penicillin, afterward combined with sulfadiazine. On April 1 many rales were heard over both lungs; the child died the same afternoon.

CASE 3.—R. W. v. L., a boy, was born spontaneously on Sept. 2, 1953, with a weight of 980 gm. The weight fell to 750 gm.; on the 28th day the birth weight was reached again. On the 51st day, the weight was 1,500 gm., and on the 66th day, 1,900 gm. On the 29th day an abscess on the right shoulder developed; it was incised on the 36th day. The pus contained Staphylococcus aureus. The child did well until the 67th day, when he became cyanotic. That day, for the first time, oxygen was administered; a flow of 4 liters a minute was given for several hours; it was afterward reduced to 1 liter a minute, and was discontinued the next day.

On the 69th day the consulting pediatrician found dullness to percussion, many crepitant rales, and diminished intensity of breath sounds over the right lung. The child was cyanotic and dyspneic. Examination of the blood showed a hemoglobin content of 54.2%; the red cell count was 3,500,000, and the white cell count was 5,600, with 44% lymphocytes, 43% polymorphonuclear cells, 3% monocytes, and 3% metamyelocytes. For three days, 50,000 units of penicillin was given three times a day, and oxygen was readministered, with a flow of 1 to 2 liters a minute, from the 69th until the 77th day. X-ray examination on the 78th day revealed questionable symptoms of pneumonia. The child died the next day.

These clinical histories show a remarkable similarity. The birth weights were lower than 1,500 gm., and the ages were between 2 and 2½ months. As all three children were treated for more than one week with oxygen administration, they probably would have been recorded in many statistics as cases of retrolental fibroplasia related to a prolonged administration of oxygen. Careful reading of the medical histories, however, reveals these facts:

1. The first infant received 8 liters a minute for four days; thereafter, during the last three weeks of life, the oxygen administration was cut down by one-half. The history in Case 2 does not mention the exact amount of oxygen given. It is noted, however, that on the 16th day before death the child did not do well without oxygen. The pediatrician, obviously, had tried to give the child as little oxygen as possible. The third infant received very little oxygen; the flow had constantly been kept below 2 liters a minute, except for the first few hours on the 67th day.

2. All three children were cyanotic and dyspneic and showed a severe anemia during the last weeks before death.

3. Anatomical examination of the eyes revealed the earliest stages of retrolental fibroplasia, which could have existed only a short time.

It seems rather certain that our three patients had been hypoxic, instead of hyperoxic, during the last period of their lives, in which the first stage of retrolental fibroplasia had developed.

INTERSTITIAL PLASMA CELL PNEUMONIA OF THE PREMATURE INFANT AND RETROLENTAL FIBROPLASIA

The general postmortem examination of our patients revealed the same remarkable similarity of findings as was shown by the clinical histories.

An interstitial subchronic pneumonia, corresponding with an advanced stage of interstitial plasma cell pneumonia of premature infants (Straub [31]), was found in all three infants. This interstitial plasma cell pneumonia of premature infants probably is an etiologic factor in a considerable number of cases of retrolental fibroplasia. It is a "new" disease, as is retrolental fibroplasia, and it also occurs predominantly in premature infants. It was described for the first time by Ammich [32] in 1938. Tobler [33] described 52 cases in 159 premature children; 66.6% of the babies with a birth weight lower than 1,500 gm. suffered from the disease, as against 60.5% of the babies with a birth weight between 1,500 and 2,000 gm. and 35.9% with a birth weight between 2,000 and 2,500 gm. Freudenberg and Tobler [34] later published new series of 65 cases in 220 premature infants, and 95 cases in 312 premature infants. The frequencies in their series for the above-mentioned birth weight groups were, respectively, 55 and 54.5%, 33 and 21.1%, and 14 and 15.7%. The incidence is inversely proportional to the birth weight, just as in retro-

lental fibroplasia. The disease is difficult to diagnose, as the onset is usually gradual and fever and cough are not present. The most important clinical symptoms are increasing tachypnea, dyspnea, and cyanosis. Physical examination often reveals minimal signs in the lungs. The disease usually develops at an age of 8 to 10 weeks. It occurs epidemically in clinics where premature infants are nursed, and it is a typical hospital disease. Freudenberg and Tobler did not find a single case in their large series which were born and nursed at home. The time of incubation is 30 to 40 days.

The disease is very probably caused by Pneumocystis carinii, an organism which for the first time was recognized as a parasite in the lungs of man by van der Meer and Brug.[35] Vaněk ‡ was the first to describe it as the causative organism in interstitial plasma cell pneumonia. Vaněk, Jírovec, and Lukeš [36] are of the opinion that Pneumocystis carinii is to be placed in the order Haplosporidia. They realize, however, that the whole order Haplosporidia is rather provisional. According to Giese,§ Pneumocystis carinii is a yeast, and he has labeled it Saccharomyces octosporus. Bauch and Ladstätter [37] agree with Giese. They succeeded in getting pure cultures of the organism, and they are of the opinion that Pneumocystis carinii belongs to the genus Candida, a yeast-like fungus. The growth of the organism could be markedly accelerated and intensified by the addition of penicillin to the medium.

The publications of Tobler and of Freudenberg and Tobler have shown that interstitial pneumonia occurs in many clinics in more than 50% of all premature infants with a birth weight below 1,500 gm., the group of infants with the highest incidence of retrolental fibroplasia. It is inevitable that many premature babies with retrolental fibroplasia have suffered from interstitial pneumonia. This is the more likely as the diagnosis is easily overlooked, and the disease is lethal in only about 20 to 30% of the cases.

The clinical, as well as the anatomical, picture of interstitial pneumonia makes it evident that the patients suffer from a severe hypoxia. The anatomical picture is characterized by an enormous thickening of the interstitial tissue of the lungs, mainly due to infiltration by lymphocytes and plasma cells. Polymorphonuclear leucocytes are scarce. The alveoli become obliterated by the increased interstitial tissue. The remaining alveolar spaces and the enlarged bronchioles are filled with a honeycomb-like, foamy material, formed by desquamated alveolar epithelium and alveolar macrophages, together with closely packed mucoid envelopes of the parasites. At autopsy the lungs, especially the dorsal parts, are seen to contain little or no air; they do not collapse, and they fill the entire pleural cavity. In fatal cases death occurs by asphyxiation. Ammich described the bronchioles as often markedly dilated, since they are shortly before death the last respiratory surface available to the infant, because of the displacement of the alveoli.

Figures 4 to 6 illustrate the lungs in my three cases. They show the advanced stage of the disease, with a marked thickening of the interstitial tissue, obliterated alveoli, and enlarged bronchioles. Figures 4 *B* and 6 *B* demonstrate the absence of polymorphonuclear leucocytes. The honeycomb-like, foamy material has partly been cleared by the alveolar macrophages. Pneumocystis organisms could not be dem-

‡ Cited by Vaněk, Jírovec, and Lukeš.[36]

§ Cited by Bauch and Ladstätter.[37]

onstrated in our cases. This may be because the organisms in this advanced stage of the disease have already been cleared by the alveolar macrophages.

The epidemic occurrence of interstitial pneumonia of premature infants reminds one of the many suggestions in the literature (reviewed by Zacharias) that retrolental fibroplasia may be due to an infection. We cite only Houlton,[38] "The appearance of numbers of cases in batches does suggest that the disease may be due to an infection, possibly by a virus," and Crosse,[39] "The grouping of cases . . . suggests the possibility of an infection, possibly in the nature of a virus." It seems plausible

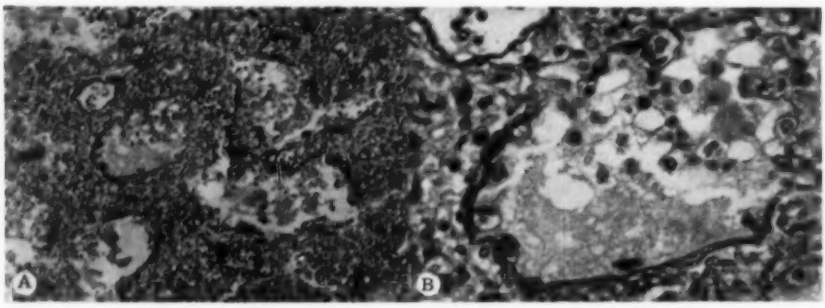

Fig. 4 (Case 1).—*A*, thickening of the pulmonary interstitial tissue with obliterated alveoli and enlarged bronchioles; × 105.

B, a bronchiole from *A*, showing honeycomb-like, foamy material within the bronchiole and absence of polymorphonuclear leucocytes; × 340.

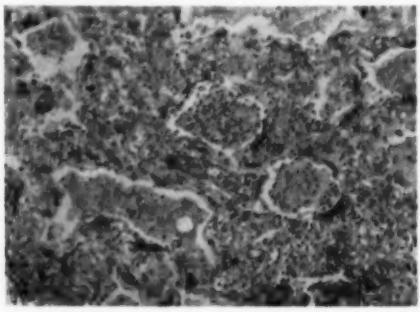

Fig. 5 (Case 2).—Thickening of the pulmonary interstitial tissue. The bronchioles and the remaining alveoli contain a foamy material with many alveolar cells and without polymorphonuclear leucocytes; × 105.

that the epidemic incidence of retrolental fibroplasia is due to the epidemic occurrence of interstitial pneumonia of premature infants. The organism "causing" retrolental fibroplasia in these epidemic cases is not present in the eyes of the patients but is found in the lungs. It causes a severe hypoxia, which is the necessary ultimate stimulus for the development of the retinal capillary proliferation that is the earliest pathological change in retrolental fibroplasia.

It may be possible that high environmental oxygen concentrations favor the development of this organism. This hypothesis receives some support from the

fact that Pneumocystis organisms are predominantly found in the lumens of the alveoli and the bronchioles and are seldom met within the interstitial tissue.

It may also be possible that the use of antibiotics favors the development of Pneumocystis. Superinfections with C. albicans (moniliasis) recently have become a well-known complication after the administration of antibiotics. Bauch and Ladstätter [37] stressed this occurrence. All their patients with interstitial plasma cell pneumonia were treated with large amounts of antibiotics. The clinical histories of our patients also show that they all received large doses of penicillin.

SUMMARY AND CONCLUSIONS

One hundred fourteen eyes of 59 premature infants who had not undergone an ophthalmological examination were anatomically investigated. The first stages of retrolental fibroplasia were found in the eyes of three children. The lungs of these three infants showed a subchronic interstitial pneumonia, corresponding to an advanced stage of interstitial plasma cell pneumonia of premature infants.

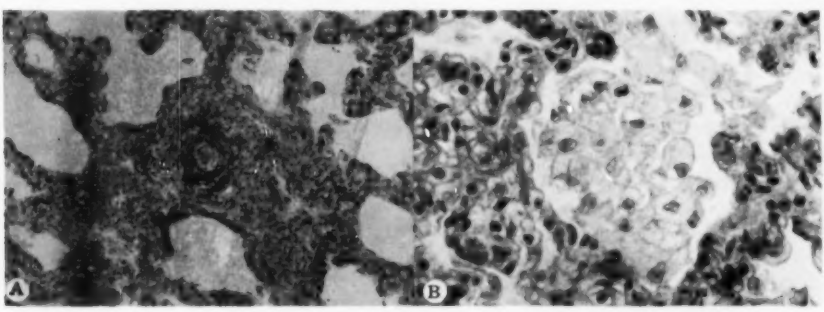

Fig. 6 (Case 3).—*A*, thickening of the interstitial tissue; × 105.
B, alveoli filled by alveolar macrophages; absence of polymorphonuclear leucocytes; × 340.

The earliest stage of retrolental fibroplasia is anatomically characterized by retinal edema and by proliferation of the capillary-forming mesenchymal cells in the nerve fiber layer.

Present knowledge of (*a*) the development of the retinal circulation, (*b*) the experiments on animals with the purpose of producing anomalies resembling retrolental fibroplasia, (*c*) the physiology of the respiratory function of the premature infant, (*d*) the incidence and the clinical picture of retrolental fibroplasia, and (*e*) the clinical histories of children who have developed this disease provide arguments for the view that retinal hypoxia is the ultimate stimulus for the proliferation of retinal capillary-forming cells in the first stage of retrolental fibroplasia.

The fact that in some clinics more than 50% of all premature babies with a birth weight below 1,500 gm. suffer from interstitial plasma cell pneumonia makes it evident that many infants with retrolental fibroplasia have suffered from this pulmonary infectious disease.

The clinical and the anatomical pictures of interstitial plasma cell pneumonia of premature infants prove that these patients are severely hypoxic. Interstitial pneumonia, therefore, is an etiologic factor in the occurrence of many cases of

retrolental fibroplasia. The epidemic incidence of interstitial plasma cell pneumonia accounts for the epidemic occurrence of retrolental fibroplasia described by many authors.

M. Straub, M.D., Pathologist of the Rotterdam Municipal Hospitals, has given particularly valuable collaboration. Professors van Bouwdijk Bastiaanse, Deelman, and Hagedoorn, and Drs. Daamen, Flieringa, Hampe, and Kloosterman provided the material and the technical facilities for this investigation.

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CHELATION OF CALCIUM WITH EDATHAMIL CALCIUM-DISODIUM IN BAND KERATOPATHY AND CORNEAL CALCIUM AFFECTIONS

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CHELATING agents (χηλή, claw) are compounds which, by forming soluble complexes with specific metals or compounds, remove them from the chemical milieu in which they occur, thus extracting, or clawing, them out.

This process can be demonstrated both in solutions and in tissues, affording us a useful tool in research and therapy. Grant [1] (1952) reported on the use of the neutral sodium salt of ethylenediamine-tetraacetic acid, edathamil calcium-disodium (EDTA *) in the chelation of calcium from corneas exhibiting band keratopathy. Significant clearing was accomplished with 0.01 or 0.05 M. solutions applied in drop fashion for about 15 minutes to the previously deepithelized corneas. The calcific component of an opacity could be removed, but actual scarring, inflammatory infiltration, or vascularization was unaffected. The visual result, therefore, depended on the proportion of these components in the corneal lesion and upon any deeper alterations present in the eye.

Band keratopathy is usually associated with long-standing uveitis, ocular degeneration, or interstitial keratitis. Hence, underlying pathologic conditions contribute to visual impairment. There is, in addition, an idiopathic form of band keratopathy, which is usually bilateral.

Another form of chemical treatment,[2] swabbing with 2% hydrochloric acid, has been used, but with undesirable side-effects. Keratectomy has been widely employed, with more favorable results. Grant's method of chelation, slightly modified as shown in the following cases, is rapid, simple, and highly effective. It should be the primary treatment in cases of band keratopathy and may make future corneal surgery unnecessary.

We have used edathamil calcium-disodium treatment only when the eye offered hope of visual improvement or when calcific corneal plaques constituted an irritant. The early treatment of lime burns of the eye with edathamil calcium-disodium, suggested by Grant, has been carried out in one case, with good results.

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*EDTA is a commercial calcium solvent marketed as Versene by Bersworth Chemical Company, Framingham, Mass.

PROCEDURE AND COMMENT

The following technique may be readily carried out as an office procedure.

1. Thoroughly cocainize the cornea. This will facilitate epithelial curettage.

2. Completely curet the epithelium up to the limbus. Failure to do so will limit the effectiveness of treatment, for edathamil calcium-disodium will not penetrate the epithelium.

3. Apply the solution of edathamil calcium-disodium in appropriate strength: 0.01 M. in light infiltrations, 0.05 M. in dense infiltrations. The solution should be warm. Instead of dropping the solution on the cornea, as advocated by Grant, we use an ordinary iontophoresis cup just fitting the cornea. The solution is poured in to a depth of several cubic centimeters, and the cup is simply held in place for 15 to 20 minutes, with occasional rotary motions to stir up the fluid. Avoid breaking the suction grip on the cornea. Fresh supplies may be instilled after sucking out the old solution in the course of the treatment. The gradual disappearance of the calcium band may actually be seen. Dense bands may require a subsequent treatment with the same or a higher strength solution—experience will indicate which. Solid plaques of calcium will not dissolve readily, but their attachment to the stroma may be loosened, permitting easy removal, en bloc, with an iris spatula. The iontophoresis cup minimizes the conjunctival chemosis which accompanies the drop treatment (and is the only unpleasant complication), insofar as it limits the contact of edathamil calcium-disodium with the cornea.

4. On completion of the corneal bath, instill an antibiotic ointment and bandage the eye tightly. Reepithelization occurs within a day or two, so that the result can be quickly assessed. The reaction of the eye is not marked, and the redness and chemosis subside quickly. Recovery was uneventful in all our patients.

Iontophoresis of edathamil calcium-disodium through the intact epithelium was ineffective.

Although we have not yet encountered a recurrence of calcium deposition, it should be a simple matter to repeat the treatment as often as necessary.

The solutions, which keep well, are prepared in the following manner:

0.01 M.: Dissolve 0.37 gm. of edathamil calcium-disodium in 100 ml. of distilled water. Add 0.10 gm. of sodium bicarbonate. Boil or autoclave the solution.

0.05 M.: Dissolve 1.85 gm. of edathamil calcium-disodium in 100 ml. of distilled water. Add 0.5 gm. of sodium bicarbonate.

REPORT OF CASES

CASE 1.—L. S., a 61-year-old white woman, came to the clinic for a routine refraction. There was no history of ocular trouble.

Visual Acuity.—O. D., 20/100, corrected to 20/60; O. S., 20/100, corrected to 20/40.

Corneas.—Bilateral, typical, band-shaped opacities encroached on the pupillary areas. Deep vascularization and infiltration were present (Fig. 1, O. S.).

Irises.—Both pupils were miotic and irregular, with posterior synechiae.

Treatment.—A solution of edathamil calcium-disodium 0.01 M. was applied to the right eye by drops for 20 minutes. There was good clearing except at the periphery, where the epithelium had not been thoroughly removed. The deep vascularization and slight stromal haze were not affected. The latter was deemed to be noncalcific. Moderate conjunctival chemosis developed. Final corrected vision was 20/25.

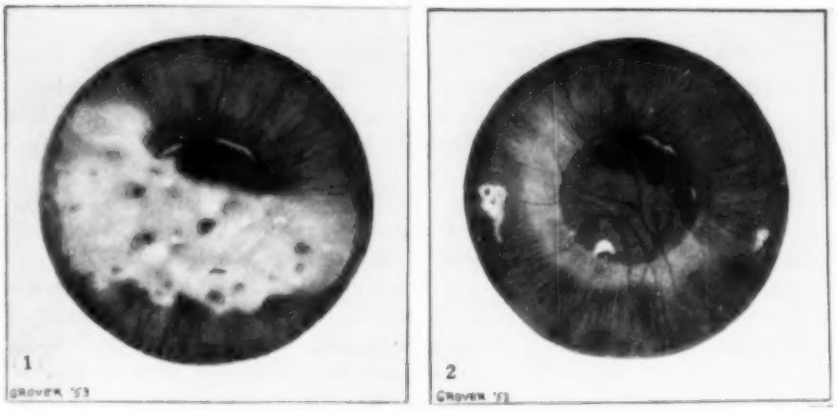

Fig. 1 (L. S.).—Artist's drawing; left eye before treatment.
Fig. 2 (L. S.).—Artist's drawing; left eye after treatment.

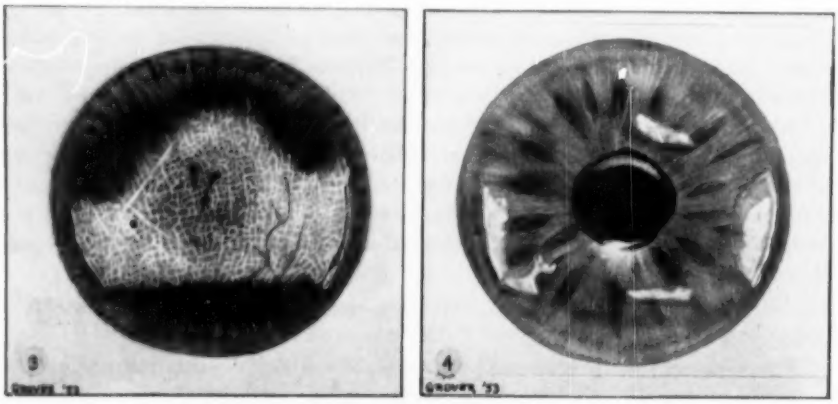

Fig. 3 (M. C.).—Artist's drawing; right eye before treatment.
Fig. 4 (M. C.).—Artist's drawing; right eye after treatment.

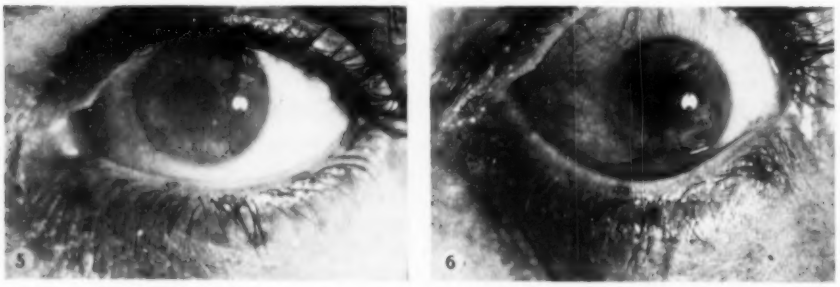

Fig. 5 (M. C.).—Left eye before treatment.
Fig. 6 (M. C.).—Left eye after treatment.

Iontophoresis with 0.01 M. edathamil calcium-disodium was attempted through the intact cornea of the left eye, without benefit. Several days later, application by means of the iontophoresis cup to the curetted cornea produced satisfactory clearing, with residual haze (Fig. 2). Chemosis was minimal. Final corrected vision was 20/25.

A follow-up nine months later revealed no recurrence.

CASE 2.—M. C., a 49-year-old white man, had noted grayish clouding of both corneas for three years. His only ocular complaint had been high myopia. He appeared markedly apathetic.

Visual Acuity.—O. U., 20/300.

Corneas.—Dense, band-shaped infiltrates covered the pupillary areas of both eyes (Fig. 3, O. D.; Fig. 5, O. S.).

Treatment.—A 0.01 M. solution of edathamil calcium-disodium was applied to the left cornea by drops. After 20 minutes the band revealed many fissures and cracks, resembling a thawing ice field. Considerable chemosis developed, lasting several days. The patient was then given a corneal bath by iontophoresis cup of 0.05 M. edathamil calcium-disodium for 15 minutes. No

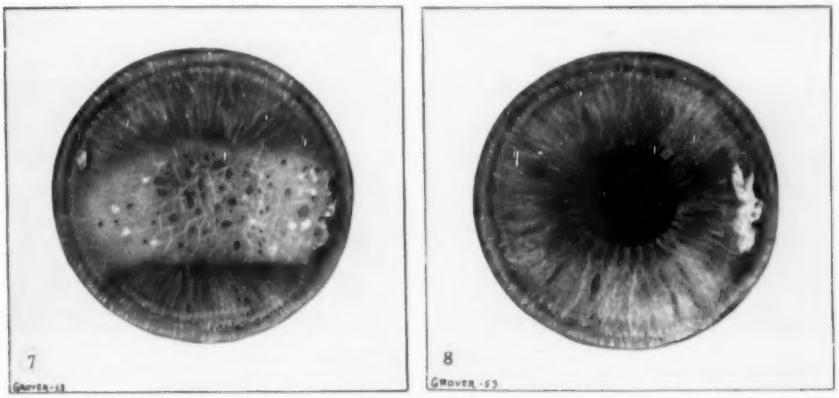

Fig. 7 (H. S.).—Artist's drawing; right eye before treatment.
Fig. 8 (H. S.).—Artist's drawing; right eye after treatment.

edema resulted, and the band dissolved, revealing a pupil bound down with synechiae. These were broken by mydriatics. Final corrected vision was 20/20 (Fig. 4).

A single corneal bath of 0.05 M. edathamil calcium-disodium was given to the right eye, with marked clearing of the band, although not as complete as in the left eye (Fig. 6). Chemosis was minimal. Posterior synechiae were partly broken. Final visual acuity was 20/50. The increase in visual acuity was accompanied by a remarkable personality change, the patient becoming alert and active.

CASE 3.—H. S., a 23-year-old white woman, stated that she had had a "corneal band" in the right eye for an indefinite period. High myopia was present, but there was no history of uveitis (Fig. 7).

Visual Acuity.—O. D., 15/200; O. S., 20/200.

Cornea.—The right eye contained a typical band opacity.

Therapy.—A corneal bath of 0.01 M. edathamil calcium-disodium was applied by cup. There was marked clearing of the cornea, but a cataract was present behind (Fig. 8).

CASE 4.—E. L., a 51-year-old white woman, had an old corneal scar, O. D., the result of a cat scratch at the age of 2 years (Fig. 9). Recently she had had persistent foreign body sensation.

Visual Acuity.—O. D., hand movements; O. S., 20/20.

Cornea.—The right eye contained a dense central scar, obstructing the pupillary area. There was deep vascularization and an adherent leukoma at 6 and 12 o'clock. In the center of the scar, occupying about one-third of the anterior corneal thickness, was a dense calcium plaque. The epithelium over and adjacent to the plaque was eroded. Cortisone and antibiotic therapy did not improve the condition.

Therapy.—A 0.05 M. solution of edathamil calcium-disodium was applied for 15 minutes by corneal bath. The plaque was too dense to be dissolved, but its attachment to the underlying stroma was loosened, so that it was lifted out effortlessly with an iris spatula. There was slight oozing of blood in the bed, but reepithelization was prompt and in two weeks the patient was completely comfortable (Fig. 10). Vision was unaffected.

Case 5.—E. W., a 36-year-old white woman, had an attack of uveitis, O. D., in 1948, with development of complicated cataract and posterior synechiae. In 1951 band keratopathy appeared, occupying the lower one-third of the cornea.

Visual acuity was light perception, O. D., and 20/15, O. S.

A 0.01 M. solution of edathamil calcium-disodium was applied for 15 minutes, with complete dissolution of the calcium. A follow-up six months later revealed a questionable return of calcium in a tiny patch.

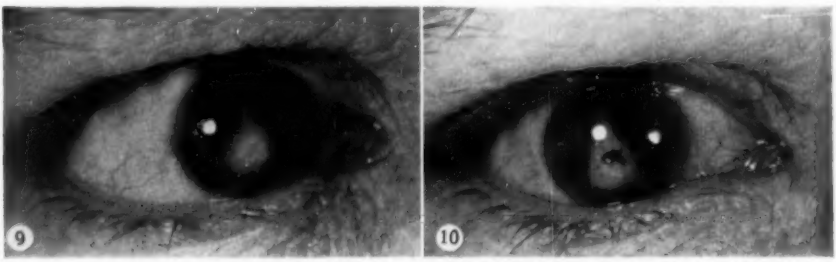

Fig. 9 (E. L.).—Right eye before treatment.
Fig. 10 (E. L.).—Right eye after treatment.

Case 6.—H. M., a 55-year-old white man, had a superficial, dense, white, circumscribed opacity at the center of the left cornea. The stroma was thickened centrally, and there was a pebble-like irregularity of the epithelium.

A 0.01 M. solution of edathamil calcium-disodium was applied, with very slight change in the appearance. The opacity was probably nearly all connective tissue.

Case 7.—R. M., a 58-year-old white man, had suffered an iodine burn of the right cornea 18 years before, with subsequent dense opacification.

Visual Acuity.—O. D., hand movements; O. S., 20/20.

Cornea.—There was diffuse opacity of the cornea (O. D.) with clumps of calcium granules in the center. Both deep and superficial vascularization was present.

Therapy.—A 0.01 M. solution of edathamil calcium-disodium was applied by corneal bath, with consequent removal of the granules. The scar tissue was unaffected. Vision was unimproved.

Case 8.—A. I., a 55-year-old white man, plasterer, splashed lime into the left eye. When he was seen several hours later, the corneal epithelium was completely denuded. The eye was irrigated copiously with isotonic saline, and particles of lime were picked out. It was then irrigated with 0.01 M. edathamil calcium-disodium. Cortisone ointment was instilled and the eye patched. In several days there was marked reepithelization of the cornea, and in a week almost complete healing. There were no sequelae.

SUMMARY AND CONCLUSIONS

Extraction of calcium from the cornea by means of the solvent edathamil calcium-disodium (EDTA), as described by Grant (with slightly modified technique), is simple, rapid, and effective. It may be carried out as an office procedure.

A 0.01 or 0.05 M. solution of edathamil calcium-disodium is applied as a corneal bath by an iontophoresis cup to the deepithelized cornea for 15 to 20 minutes. This application suffices in most cases to produce complete clearing of the calcific component of band keratopathy. There is no effect on scar tissue.

If the underlying ocular structures are intact, marked visual improvement may be expected.

A series of 10 eyes are presented, with band keratopathy or other calcium affections of the cornea, in which this treatment was effective.

Calcium chelation should be the primary treatment for band keratopathy and irritating calcium infiltrations of the cornea. It is also of value in the early treatment of lime burns of the cornea and conjunctiva.

ADDENDUM

Satisfactory corneal cups may be improvised from test tubes or small medicine bottles.

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CATARACT AND RETINAL DETACHMENT ASSOCIATED WITH ATOPIC DERMATITIS

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THE THREE diseases with important skin manifestations in which cataracts occur with some frequency are Rothmund's syndrome, Werner's syndrome, and atopic dermatitis. According to Buschke,[1] cataract may be seen in rare cases of keratosis follicularis, telangiectases, and developmental anomalies of the hair. In a comprehensive study of Werner's syndrome and Rothmund's syndrome, Thannhauser [2] pointed out their differences and showed that cataract is an integral part of each entity. He also stated that myotonia dystrophica, in which cataracts are frequently found, may at times resemble Werner's syndrome. These syndromes are rare and heredofamilial. Atopic dermatitis, however, is a relatively common skin disease, and the presence of cataract adds a most distressing complication to a disease which is notoriously difficult to manage. In addition to the increased physical disability, a psychic trauma is heaped upon a personality ill equipped to bear it.

Androgsky [3] in 1914 reported the first four cases of cataract associated with atopic dermatitis, and in 1921 Davis [4] recorded the first case in this country, that of a 15-year-old girl with bilateral cataract. Since the original description, infrequent reports of additional cases have been recorded, mostly in the dermatological and ophthalmological literature. Sack [5] in 1947 reviewed the literature and found a total of 27 cases in the European journals, 33 in journals of the United States, and 1 in Australian records. Coles and Laval [6] stated in 1952 that the number of reported cases had risen to 63. In view of the few cases recorded and the opinions expressed by a number of authors, cataract has been considered rare in atopic dermatitis. In 1936 Brunsting,[7] in a comprehensive review, found cataract present in 10 of the 101 cases. However, these cases were selected and were not used to determine the incidence of atopic cataract. Later, in 1943, Brunsting,[8] in his comments on a case, stated that he had observed 40 cases of atopic dermatitis complicated by cataract formation. In 1950 Cowan and Klauder,[9] in a control study of 100 patients, all under the age of 40, found 8% to have lenticular changes which the authors believed characteristic of atopic cataract. In only 3%, however, was there impairment of vision. A search of the records of the Presbyterian Hospital since 1932 revealed eight cases of atopic cataract in which vision was reduced. In all these cases the cataract was bilateral.

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The cataract generally appears in the adolescent or early postadolescent period, after the dermatitis has become well established, but a few cases have occurred after the age of 40 or in the preadolescent period. There is no striking predilection for either sex, though of the 61 previously reported cases reviewed by Sack,[5] 35 were of males. In this same group 50 patients had bilateral cataract. According to Sack, the average time interval between the first appearance of the dermatitis and the development of the cataract is 15 years. The lenticular changes are often rapidly progressive once they begin, and a mature cataract may be formed within a few months. Milner [10] has described a case in which while the patient was under his observation a cataract developed which within one week reduced vision from 6/6 to 6/18.

According to Beetham,[11] who has reported on the study of 18 eyes with atopic cataract, there are two types of lenticular changes. The commonest begins at the posterior pole of the lens with iridescent crystals, vacuoles and precipitates, and small, punctate opacities. There is peripheral spread, with similar changes soon noted in the anterior cortex just beneath the capsule. The changes progress to cloudiness of the entire cortex, and a homogeneous mature cataract is formed. The second type is described as a dense, irregular plaque of opacity in the anterior cortex immediately beneath the capsule, with occasionally some wrinkling and elevation of the normal capsule. This type also progresses to involve the whole lens.

REPORT OF CASES

Case 1.—W. H., a white, single man of 23, was first seen in June, 1953, for a pruritic, scaly, moderately inflammatory, somewhat lichenified eruption of the hands, arms, face, and neck. During infancy he had an eczematous eruption, which lasted only a few months. At the age of 16, while attending a rather strict boarding school, he suffered from nervous indigestion with nausea and vomiting. During this time his skin eruption recurred and, except for occasional remissions, has continued to cause much discomfort. In December, 1950, intradermal tests by Dr. Clifford G. Weston showed positive reactions to various dusts, feathers, ragweeds, molds, and foods. Elimination diets were thought to give some improvement, but the patient was not impressed by their effect. In January, 1951, penicillin was given for acute gingivitis, and a severe generalized eczematous eruption followed, requiring hospitalization. During the winter of 1951 the patient spent some time in Texas, with much improvement, but became worse when he returned home. In 1951 he received four x-ray treatments to the affected skin.

In February, 1951, examination by Dr. Hobart McV. Agnew showed vision of 20/20 in each eye. On July 7, 1951, vision in the right eye was reduced to 20/50 and by Aug. 8, 1951, the patient could distinguish hand movements only in the right eye, and vision in the left eye was reduced to 20/40. On April 29, 1952, an intracapsular extraction of the lens was performed by Dr. Agnew, and corrected vision in this eye has remained 20/20. The cataract in the left eye developed more slowly, and by January, 1954, was approaching maturity (Fig. 1).

No family history of allergy could be elicited, and the patient's past history revealed no other allergies or serious illnesses. Physical examination showed a tall, rather thin man, with no abnormalities except those of the skin and eyes.

Course.—During the period of observation the patient had a number of personal problems, which gave rise to considerable emotional conflict. During these episodes he was seen in a highly nervous state, with severe exacerbations of the dermatitis in which large areas of the scalp, face, neck, extremities, and trunk were inflamed, lichenified, and badly excoriated and exudative. On a number of occasions much improvement followed the resolution of a distressing problem, and on other occasions, without change in medication or daily routine, exacerbations came on the heels of an episode of emotional tension. Applied to selected areas, 2.5% hydrocortisone acetate (Cortril) ointment was very effective, and antihistamine drugs appeared to give some relief of symptoms. In order to prepare the patient for travel, he was given injections

of corticotropin, with considerable benefit. About Feb. 1, 1954, the patient moved to El Paso, Texas. Improvement was rapid, and after six weeks he reported that his skin was deeply tanned, free of eruption, and required no medication. He felt relaxed and had gained 14 lb. (6.4 kg.). In April a successful extracapsular extraction was performed on the left eye by Dr. W. G. Morrow Jr.

Case 2.—D. W., a 23-year-old white, single man, was seen in January, 1954, for a pruritic, moderately inflammatory, scaly and lichenified dermatitis of the scalp, face, neck, arms, and scrotum.

The eruption began at the age of 10 years, and the skin has remained in a chronic state of inflammation, with periods of intense itching. Severe excoriation, mostly at night, with resulting secondary infection, had been a most distressing feature. The patient recognized the importance of emotional upsets and nervous tension and believed this to be the trigger mechanism in exacerbations. He had had three years of psychotherapy and believed that this had helped to some extent, especially in partially reducing the paroxysms of excoriation. No other regimen or specific treatment, including the whole gamut of topical applications, had been of appreciable benefit.

Fig. 1 (Case 1).—Cataract in the left eye and some lichenification of the skin.

In January, 1952, an opacity was first noticed in the right lens, and during the next two years the cataract developed to maturity (Fig. 2). Examination by Dr. Conrad Berens in January, 1954, showed vision in the right eye to be limited to light perception and projection. There was some atrophy of the iris, and the intraocular tension was low. Subjective tests and the reduced intraocular tension were indicative of a detached retina. The left eye showed vision of 20/20 with correction, and an incipient cortical cataract was seen in the upper half of the lens.

The family history revealed that one maternal uncle had angioneurotic edema and one cousin had rheumatic fever. One niece has atopic dermatitis; one cousin has asthma, and a sister has attacks of allergic coryza. The past history showed no hay fever or asthma, but on occasion he has had bronchitis in damp weather. Physical examination gave normal findings except for the skin and eyes.

Laboratory examinations showed the blood sugar and glucose tolerance to be normal. Intradermal tests, by Dr. Robert A. Cooke, gave positive reactions to house dust, cat dander, and Candida albicans. Other tests with the important inhalant allergens were negative, and except for chocolate a sampling of food tests gave negative results.

Course.—The patient applied 2% hydrocortisone ointment and over a period of time obtained some relief but was not able to clear up completely the areas treated. He is planning to move to the southwest part of the United States, preferably to the vicinity of El Paso.

COMMENT

The cases presented were seen in office practice and are typical of atopic dermatitis with cataract. In each case exacerbations of the dermatitis were closely correlated with periods of emotional stress. In Case 2 the additional complication of detached retina in the right eye was present. Detached retina may occur with atopic cataract before surgery is performed, or it may follow cataract extraction. MacDannald [12] and other authors have indicated that surgery for this type of cataract entails no particular hazard. However, Brunsting advised extracapsular extraction because of risk of rupturing the fragile capsule, and Coles and Laval [6]

Fig. 2.—Cataract in the right eye and sparsity of the lateral portion of the eyebrow.

empasized the dangers inherent in surgery for this type of cataract. The latter authors have collected reports of 21 eyes with atopic cataract which became blind or were enucleated as a result of complications, the principal cause being detached retina. In addition, they present a case in which bilateral cataract extraction was followed by detached retina, leaving the patient totally blind. In this case, as in that of Cordes and Cordero-Moreno,[13] the patient vigorously rubbed and slapped the face and eyelids to allay itching. While such trauma may be only a possible contributing cause of retinal detachment, it may be a direct factor in postoperative infection, as in the case of panophthalmitis described by Thompson.[14] Therefore, it is important to place the skin in the best condition possible before undertaking surgery for atopic cataract. Thompson [14] listed a number of points deemed important in the preoperative and postoperative care of these patients and stressed the need for proper preparation from an emotional standpoint. Adequate sedation, control of secondary infection on the skin, and the wise choice of topical applications are all important.

The true incidence of cataract associated with atopic dermatitis is difficult to determine unless patients are observed over a long period of time with slit-lamp examinations. It is probable that some of the early minimal lenticular changes thought to indicate atopic cataract may not necessarily progress to impair vision, and it is believed that the chance of reduced vision from cataract in atopic dermatitis is actually less than the 3% indicated in the report of Cowan and Klauder. However, ophthalmological examination is valuable in these cases, and if progressive lenticular changes are found, there would be additional reason for the patient to seek an environment favorable to the skin in the hope of retarding the cataract formation.

The etiology of this type of cataract remains obscure, and there is no known method of prevention. Numerous theories have been advocated to explain the lenticular changes, such as nutritional, hormonal, or electrolytic imbalance, but none is supported by proof. The theory that the x-ray therapy which some of these patients have received is the cause of the cataracts is untenable when one considers that common skin diseases, such as acne, often receive x-ray therapy and are not attended by cataract formation. It seems most logical to believe that in the atopic state when the skin reacts as a shock organ, the lens, which is also ectodermal in origin, can do likewise. However, increased capsular permeability in these cases has not been proved experimentally, and it is not known whether the allergenic properties of the lenticular alpha globulin are related to factors present in the skin or aqueous. The retinal detachments have been explained on the basis of retinal edema secondary to vascular changes, but Coles and Laval believe the primary cause to be a diseased vitreous which fails to hold the retina firmly in place.

Many observers since the time of Besnier have recognized the role of the psyche in the pathogenesis of atopic dermatitis in adolescents and adults. These patients are frequently intelligent, sensitive, poorly adjusted persons, who are inclined to be perfectionists and who react adversely to the emotional stress of competitive life. Although there is often a strong personal and familial allergic background and multiple hypersensitivities are frequently demonstrable, there is poor correlation between the results of cutaneous tests and the production of symptoms in the individual case. It seems reasonable to consider atopic dermatitis to be, at least in part, a disease of stress in a diathetic person. Study in line with the broad concepts of the general adaptation syndrome of Selye may help to clarify the role of various systems, such as the autonomic nervous system, in the hypersensitivity of these patients.

In addition to the patient whose case is reported, we have observed a number of patients who after years of unsuccessful medical treatment have moved to the vicinity of El Paso, Texas. There they became asymptomatic, but on brief visits to their old environments the dermatitis returned temporarily. Frequent sun bathing has been considered necessary, and some mild recurrences have appeared during the winter months. Some of these patients left personal problems behind, but factors such as increased exposure to sunlight, the arid atmosphere, and changes in exposure to allergens must be studied to explain the remission of symptoms.

SUMMARY

Two cases of bilateral cataract associated with atopic dermatitis are presented, one with an additional complication of detached retina. This type of cataract is generally bilateral and may develop rapidly, as in Case 1, and reach maturity within

a few months. Ophthalmological examination is advisable in order to detect early lenticular changes. The great tragedy of permanent blindness at an early age is sometimes the result of retinal detachment or other postoperative complications. Maximum improvement of the skin should be obtained before operation is performed. The sustained improvement of patients with atopic dermatitis after moving to a particular warm, arid environment deserves further study.

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DIVERGENCE INSUFFICIENCY

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THERE seems to be an increase in the incidence of divergence insufficiency. This may be due to stimulated interest and improving diagnostic acumen. However, it may be the result of increase in certain types of brain stem involvement produced by automobile injuries, virus encephalitis, and vascular lesions occurring in advanced years—very different causes, which have furnished a number of the cases in the recent series we have studied.

Another reason for this discussion of this subject is the revelation of investigators who have rather definitely proved that divergence function is an active process, and not merely the result of convergence relaxation or the return to the rest state of ocular muscle balance. Adler and his co-workers demonstrated that an actual change, an increase in electrical potential, took place in the lateral rectus muscle when the eye was moved from a convergent to a less convergent position.[1]

A study of the cases herein reported suggests reason for further discussion of the question as to whether there is a divergence center, such as has been established in Perlia's nucleus for convergence. For the present, one had best replace the idea of a center for divergence by a more extensive "neural arc" or "relay section," through which are transmitted selected stimuli.

In the study of the central nervous system one must always consider structure and function together. One does not exist without its complement. The so-called "center," in effect a relay station, screens and selects certain stimuli to pass along the arc to the next station. Other sensations are ignored or discarded. The stimuli refused further projection may be too weak to make proper impression, or may be of different variety, and not acceptable.

This brings us to the consideration of selectivity of cells and their projections in response to certain physiologic impulses or noxious influences.

This property of selectivity is definitely present. One can administer certain of the eight aminoquinoline drugs and find that their toxic effect destroys only certain cells of the oculomotor nucleus. If another drug of the same group is given, so similar that merely a radical is changed in its position, other cells of the oculomotor nuclei are affected.

Pressure itself without direct damage, possibly by the action of anoxia and anemia, may also impair the function of cells or their processes, the selected effect being due either to varied sensitivity or to greater or less exposure. This is seen in the cases of herniation of the uncus of the temporal lobe through the hiatus

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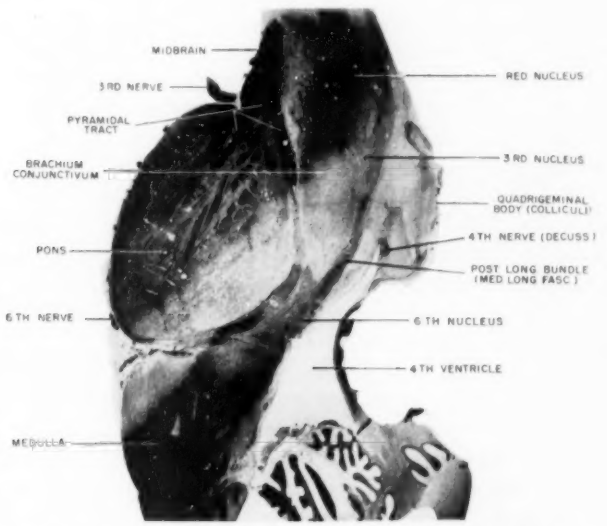
of the cerebellar tentorium. The oculomotor nerve is pressed between the herniating structure and the rigid rim of the tentorium. Because of greater sensitivity or greater exposure, a dilated pupil is produced. Ptosis and extraocular paralysis do not appear until late, if at all.

To apply selectivity to this presentation is not difficult, as clinical material, although not abundant, definitely shows the same pattern.

The area with which we are principally concerned is the brain stem, particularly the pons and the midbrain, which contain the oculomotor, trochlear, and abducens nuclei and their associated or connecting fibers and tracts. The chief association tract is the posterior longitudinal bundle, or median longitudinal fasciculus, or, more succinctly termed, the final common pathway along which pass stimuli, reflex for the most part, serving at least eight reactions. Possibly all of these are concerned with eye movements.



Lateral section of brain stem through posterior longitudinal bundle.

As the posterior longitudinal bundle is the first collection of longitudinal fibers to form in the brain stem in both phylogenetic and ontogenetic development, selectivity of function obviously is associated with time of accretion of new fibers and positioning of fiber bundles which comprise the entire tract.

There are many nuclei scattered throughout the reticular substance of pons and midbrain from which fibers course, chiefly longitudinally. Some of these may be concerned with eye movements. At the present time the function of these cells, or collection of cells with their processes, is not definitely known.

Stimulation for voluntary eye movements initiated in the frontal lobe, directed by higher, so-called psychic areas, passes to the oculogyric nuclei through the aberrant pyramidal, or corticobulbar, tract to merge in the posterior longitudinal bundle. A lesion of this system in this so-called internuclear area produces incoordination or dissociation of eye movements.

Proprioceptive fibers from all parts of the body and from the vestibular nuclei also merge in the posterior longitudinal bundle. A lesion in this section produces incoordination of eye movements and influences nystagmus when present. These systems convey ascending stimuli in the posterior longitudinal bundle.

Another system, part voluntary, part reflex, which may be called involuntary, is the visuomotor, the occipital-mesencephalic, tract, which conveys impulses, originating in the visual system, to the extraocular and intraocular muscles. This system adjusts the eyes upon the object of regard through centering, fixing, and following, as well as focusing—a vergence function. Clinical studies supplementing animal experimentation indicate that the stimulus serving these functions passes in its final section down the posterior longitudinal bundle. A lesion in the lower midbrain below the convergence control, or upper pons, produces convergence through curtailment of bilateral abducens action.

A lesion in this section, if small and isolated, may involve fibers serving divergence, at first weakening that function, and with increasing damage paralyzing it. If the lesion expands, it may include voluntary corticobulbar fibers, resulting in dissociation of eye movements, or vestibular fibers affecting, probably suppressing, pathogenic or induced nystagmus. The lesion may develop to implicate the abducens nuclei in producing an infranuclear noncomitant abducens paralysis. It may extend upward to produce oculomotor paralysis, paralysis of convergence, loss of accommodation, and paralysis of vertical gaze.

The patient with divergence insufficiency presents himself complaining of double vision for distance, which increases with distance. He usually volunteers the information that he has no trouble in reading or close use of his eyes.

Histories reveal the possibility of a number of etiologic factors. Recent trauma or infection is a most frequent cause, an automobile accident or fall, or a virus infection, usually beginning with the respiratory system and developing meningitic or encephalitic symptoms. Vascular lesions produce this condition in the aged and sclerotic, as a result of thrombosis and degeneration. Tumors, either with direct destruction or with secondary compression, are a cause; in fact, the compression is of such importance that several authors * have presented a syndrome which includes divergence paralysis, headache, and papilledema from increased intracranial pressure. Roentgenologic studies reveal obstructive hydrocephalus with occlusion or dilatation of the fourth ventricle or lower portion of the aqueduct of Sylvius. Other causes are seen and have been reported in the production of divergence insufficiency. They include multiple sclerosis, syphilis, diphtheria, chorea, lead poisoning, alcohol, and other toxic agents. Anoxia has been shown experimentally to produce divergence insufficiency. Functional cases have been reported due to hysteria and neurasthenia.

The only subjective symptom, the sudden onset of diplopia at distance, has been mentioned. This diplopia increases with far distance and upon looking down. It may decrease upon looking upward and laterally unless the lesion is low in the pons and implicates a part of one or both abducens nuclei. Single vision is found within close range of 1 or 2 ft. (30 or 60 cm.). Other subjective symptoms may arise as adjacent tracts or nuclei are implicated through expansion of the lesion.

* References 2 and 8.

Objective symptoms, unless extension occurs, are constant. The deviation of the eyes is not evident upon casual inspection and requires examination of all types of ocular muscular action. The usual Maddox rod, screen, and prism, and Lancaster tests reveal what appears to be moderate homonymous concomitant convergent heterophoria. However, in these cases there is lacking the usual control of fusion through innervational adjustment. Without this reserve threshold, the condition asserts itself as manifest heterotropia. Divergence tests with the rotary prism or the major amblyoscope show divergence action to be absent or greatly reduced.

Versions, with minor reservations, are essentially normal. If the patient is asked to follow an object with his eyes 40 or 45 degrees to the side in horizontal version and the adducting eye is quickly covered, the abducting eye will be seen to jump to greater abduction. This version adjustment, equal on the two sides, is evidence of vergence weakness, at the same time demonstrating slight version weakness on extensive excursion, and normal duction. In these cases convergence, accommodation, and pupil reflexes are normal unless the upper midbrain is implicated by expansion of the lesion.

In differential diagnosis one must consider the following possibilities:

1. Spasm of convergence in which diplopia increases at near and is reduced by cycloplegic drugs.

2. Bilateral or unilateral abducens paralysis in which the diplopia is noncomitant, increasing when the eye is turned in the direction of the involved muscle.

3. Hemorrhagic polioencephalitis superior of Wernicke, and other types of encephalitis, in which there is extraocular or intraocular muscular incoordination, bilateral or unilateral.

4. Dissociation of eye movements, including (*a*) anterior internuclear ophthalmoplegia of Lhermitte, with paralysis of conjugate lateral gaze, mesial movements failing, with preservation of convergence, and (*b*) syndrome of Raymond Cestan, with dissociation of conjugate lateral gaze together with contralateral hemiplegia.

5. Myasthenia gravis, which is relieved by neostigmine and which is intermittent in occurrence and inconstant in amount.

6. Thyrotoxic ophthalmoplegia, which at present is considered to be produced by change in the extraocular muscles.

7. Various types of ocular myopathies, hereditary and acquired.

Prognosis and treatment depend upon the type of lesion. Obviously, tumors, infection, and vascular lesions must receive appropriate treatment. The response determines the prognosis.

We have found that Grab-Front prisms, based out, serve very well and relieve the patient of diplopia or the need for occluding one eye. The Grab-Front prisms are removed usually when reading. If the condition is prolonged, as is found in many cases of trauma, glasses may be made with prisms in the upper part, for distance only. If the condition persists, operation on both lateral rectus muscles may be considered. Advancing, tucking, or resection may be the choice of the surgeon. Satisfactory results are usually obtained.

REPORT OF CASES

Abstracts are herewith presented of pertinent features in the case histories of patients with divergence insufficiency, having the most usual causes.

1. *Inflammatory, infectious: virus encephalitis.*

R. H., a woman aged 21, had virus infection, beginning two months prior to eye examination, affecting the respiratory system, with symptoms, a week later, of headache and stiff neck, then

infranuclear right facial paralysis. These symptoms subsided, and convalescence was uneventful until double vision for distance was noticed. Diplopia persisted, and the patient was referred for eye examination.

Vision was 20/25, right eye, and 20/20, left eye, uncorrected. Visual fields were normal. Optokinetic nystagmus tests produced a normal reaction. A neostigmine test for myasthenia gravis was negative. Serological tests were negative. The patient had been seen by a neurologist, who summarized his examination by the information, "focal encephalitis or peripheral cranial neuritis or neuropathy, possibly due to virus."

Homonymous esotropia was present for distance and remained unchanged with version. With extreme version nystagmus was elicited in the direction of deviation. Slight nystagmus was present on looking upward; none on looking downward. It was thought that this was a fatigue or exertion type of nystagmus or nystagmoid movement. Vision within 18 in. (46 cm.) was single. No muscle weakness was found with duction tests.

Patching one eye alternatively was advised. In three weeks the diplopia had disappeared. Medication for the virus infection was chlortetracycline (Aureomycin) and thiamine chloride.

2. Brain stem injury.

M. S., a woman aged 69, was injured three weeks prior to eye examination when thrown against the windshield of an automobile. A severe headache was produced, with constant double vision for distance. Vision in the right eye was corrected to 20/30; in the left eye, to 20/20. Media and fundi were essentially normal.

Homonymous esotropia was found for distance, with slight exophoria at close, within reading range. Version at distance did not alter the diplopia. Duction tests were normal.

Grab-Front, 3-degree prisms, based out, for both eyes were prescribed to be worn over the refractive correction for distance. These glasses were worn with comfort until resection of both external rectus muscles restored single distance vision.

3. Vascular: circulatory insufficiency, vascular sclerosis, thrombosis.

E. R., a woman aged 81, complained of intermittent double vision for distance, which during the week prior to the first examination had become constant. Three years previously double vision developed for distance and lasted for two months, then gradually subsided. There was no illness or injury at that time. The second episode occurred one year later and lasted three months. The third attack of diplopia began four months prior to examination and continued with remissions until the time of the examination. General physical examination was essentially normal. General vascular sclerosis was evident. Sclerosis was rather marked in the retinal and choroidal vessels. Vision was corrected to 20/25 and 20/20. Visual fields were normal. Optokinetic nystagmus tests gave normal response. Neostigmine tests were negative for myasthenia gravis.

Homonymous esotropia was present for distance; orthophoria, for close. Prism glasses, 3 degrees, based out, for both eyes gave comfortable single vision for distance. Prisms were removed for close.

4. Tumors, subtentorial.

M. H., a woman aged 20, complained of double vision, which had appeared gradually four weeks prior to examination. At first the diplopia was inconstant; for the last two weeks it had been continuous. Vision in each eye was 20/20. Visual fields were normal. Nystagmus was present in all positions of gaze. Diplopia was found for distance only, not varying with version. Duction tests revealed normal muscle action. Diplopia was not present at close. Optokinetic nystagmus tests were normal. The electroencephalographic report was "borderline normal EEG." Ventriculograms revealed symmetrically dilated lateral ventricles and third ventricle.

One week after the first examination of the patient attacks of vertigo occurred, followed by nausea, especially with sudden head movement. The pupils were equal and reacted normally. Bilateral papilledema appeared.

Operation revealed a tumor, roughly oval and 2.5 cm. in longest diameter, separating the cerebellar hemispheres and completely filling the fourth ventricle. The diagnosis was medulloblastoma.

The diplopia for distance, which had persisted to the time of operation, was not complained of or detected after surgery.

5. *Multiple sclerosis.*

J. H., a woman aged 55, was first seen three years prior to this report. At that time she was found to have multiple sclerosis. The discs were rather pale, although vision could be corrected to 20/30 in the right eye and 20/25 in the left. Visual fields were normal. Extraocular muscle balance was within normal limits. Nystagmus, scanning speech, and spastic paraplegia involving the lower extremities were present.

A year and a half later the patient was seen because she complained of diplopia for distance with single vision at close. She was found to have homonymous esotropia for distance, not changing in amount with version. Duction responses were normal. Nystagmus was present in all positions.

Prisms, 4 degrees, based out, were prescribed, for both eyes. These prisms have been worn for six months, for distance, over the glasses with comfort.

SUMMARY

The subjective symptom of divergence insufficiency is sudden appearance of double vision for distance. Single vision remains for close.

The cause of diplopia is not obvious upon casual inspection and requires examination of all types of muscle action. The usual tests reveal what appears to be moderate concomitant esophoria for distance, but, because of disturbance of innervation, homonymous diplopia is present. The condition in reality is manifest bilateral convergent heterotropia.

Versions, with minor reservations, are essentially normal. Ductions and convergence are normal. Divergence is absent or greatly reduced.

This condition is usually the result of varied pathologic conditions involving the brain stem, particularly the midbrain and pons.

Prognosis depends upon etiology.

Ophthalmologic treatment consists of prisms or occlusion of one eye. Surgery of the lateral rectus muscles may be resorted to if diplopia persists.

Union Trust Building (2).

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CONGENITAL ENTROPION AND TETRASTICHIASIS OF UPPER LIDS, PALPEBRAL HYPERPIGMENTATION, AND MENTAL DEFICIENCY

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IN HER WORK on congenital and developmental anomalies, Mann [1] has noted that congenital entropion is exceedingly rare. Congenital entropion of the upper lid may be said to be extremely rare. The first case reported was that of von Ammon [2] in 1841. He published illustrations of the eyes of a 3-year-old girl with multiple congenital anomalies. The right eye showed ectropion of the lower lid, entropion of the upper lid, and dyscoria. The left eye showed entropion of both lids. No further observations on the condition of the tarsus were made. The next case reported was Wilde's,[3] in 1844, cited by Harlan,[4] in which only the right upper lid was involved. In 1894 Lippincott [5] described a bilateral case. Harlan,[4] in 1895, presented a case in which the tarsus of both upper lids consisted of narrow, slightly thickened bands. Upon eversion of the lids grooves could be found in the conjunctiva, and a probe could be passed easily via the grooves between the conjunctiva and the tarsus, since the usual adhesions between the two were absent. There was entropion of the whole upper lid in the right eye, but only of the nasal one-third in the left eye, the tarsus being normal lateral to this. De Schweinitz,* in commenting on this case, described an almost identical case in which the entropion involved the nasal half of the right upper lid and the lateral two-thirds of the left upper lid. In this case conjunctivitis was observed on the third day of life, persisting for about one month, yet yielding no organisms on culture.

In 1934 Chow [6] reported a 5-week-old child with multiple congenital anomalies. The right eye could not be opened entirely until one week after birth, and from that time the upper lid remained turned inward, so that the cilia scratched the cornea. Arkin,[7] in 1935, described an interesting case in an 8-month-old boy with other congenital anomalies. In addition to the entropion of both upper lids, there was slight microphthalmos, inferolateral obliquity of the orbital rims, epiblepharon, epicanthus, occlusion of the nasolacrimal ducts, cardiac disease, a quadrantal skull with hypertelorism, brachysyndactyly of the fourth and fifth fingers, bilateral inguinal hernia, enlarged ears, and left equinovarus. In this case the tarsi were considered normal.

Redslob,[8] in 1947, described a case involving the right upper lid, especially noteworthy because the tarsus was found to be greatly thickened, owing to a second row of Meibomian glands. Duke-Elder [9] mentions two familial cases characterized by complete absence of the tarsus.

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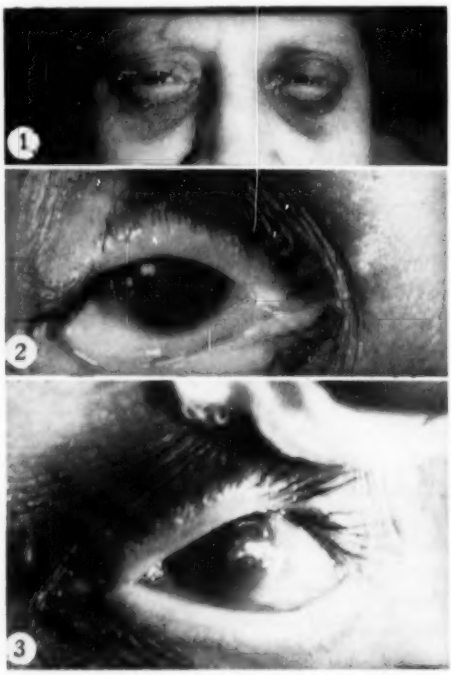
* de Schweinitz, G., in discussion on Harlan.[4]

REPORT OF A CASE

We wish to present the case of a 32-year-old Negro woman with entropion and trichiasis of the lateral half of both upper lids. Other abnormalities noted were tetrastichiasis, hyperpigmentation of the palpebral regions, and low intelligence. That the condition was congenital was verified by an older sister, who had observed the patient from the time of birth. The sister had no entropion, but she also had tetrastichiasis and trichiasis. In addition, she had a vestigial accessory finger at the lateral margin of each hand. A third sister, without any lid abnormalities, also had polydactyly.

There were only two points of interest in the general history. One was the detection of "bad blood" on the patient's arrival in New York from the South, in 1938. It could not be ascertained whether or not the positive Wassermann reaction was due to congenital syphilis



Figs. 1-3.—*1*, from a Kodachrome, illustrating hyperpigmentation of the palpebral areas and marked restriction of opening of the palpebral fissure. *2*, right eye, from a Kodachrome, showing the entropion and lateral pannus. *3*, left eye, from a Kodachrome, showing the entropion and lateral pannus.

or to the acquired form. However, she bore none of the stigmata of prenatal syphilis. This finding is of interest because of the theoretical possibility that the entropion was due to a prenatal syphilitic tarsitis. The other point was the curious nature of the hyperpigmentation of the palpebral areas. This can readily be observed in Figure 1, the appearance being somewhat raccoon-like. This pigmentation, as might be expected, waxed and waned with each menstrual cycle and became much more marked during pregnancy.

General examination revealed no abnormalities apart from her eyes. Results of psychiatric examination indicated that she was a rather high-grade mental defective.

Ocular examination revealed the aforementioned hyperpigmentation. The palpebral fissures were very narrow, being about 2 to 4 mm. in width, as may be seen in Figure 1. The patient

appeared to suffer from severe photophobia, and examination was difficult. However, she stated that this condition caused her no pain, even though the lashes appeared to rub constantly against the corneae. Tetracaine reduced the photophobia only slightly. True ptosis was not present, since topical anesthesia and patient coaxing induced her to open her eyes to 10 mm. There was entropion of the lateral half of each upper lid for 13 to 14 mm., the length of the palpebral fissures being 27 mm. Tetrastichiasis was present the length of both upper lids, and trichiasis, for the lateral half of each upper lid. There was one row of Meibomian orifices in each lid. The irritation of the cornea by the cilia had produced scarring of the lateral one-third of the right cornea and the lateral one-half of the left. Biomicroscopy showed superficial keratitis with considerable vascularity, but no staining. The involved areas were slightly raised. They can be seen in the photographs (Figs. 2 and 3) and in the artist's drawings (Figs. 4 and 5). Corneal sensitivity was absent over the diseased areas but was normal in the uninvolved cornea. In the conjunctiva, lateral to each cornea was a marked accentuation of the Negroid pigmentary corona radiata. On eversion of the lids (achieved with considerable difficulty), the normal concavity of the tarsal plates was seen to be exaggerated in the vertical meridian laterally. The palpebral conjunctival surfaces showed diffuse scarring, such as occurs after trachoma. However, the diagnosis of trachoma was considered unlikely because of the history, the clinical course, and the lateral position of the pannus.

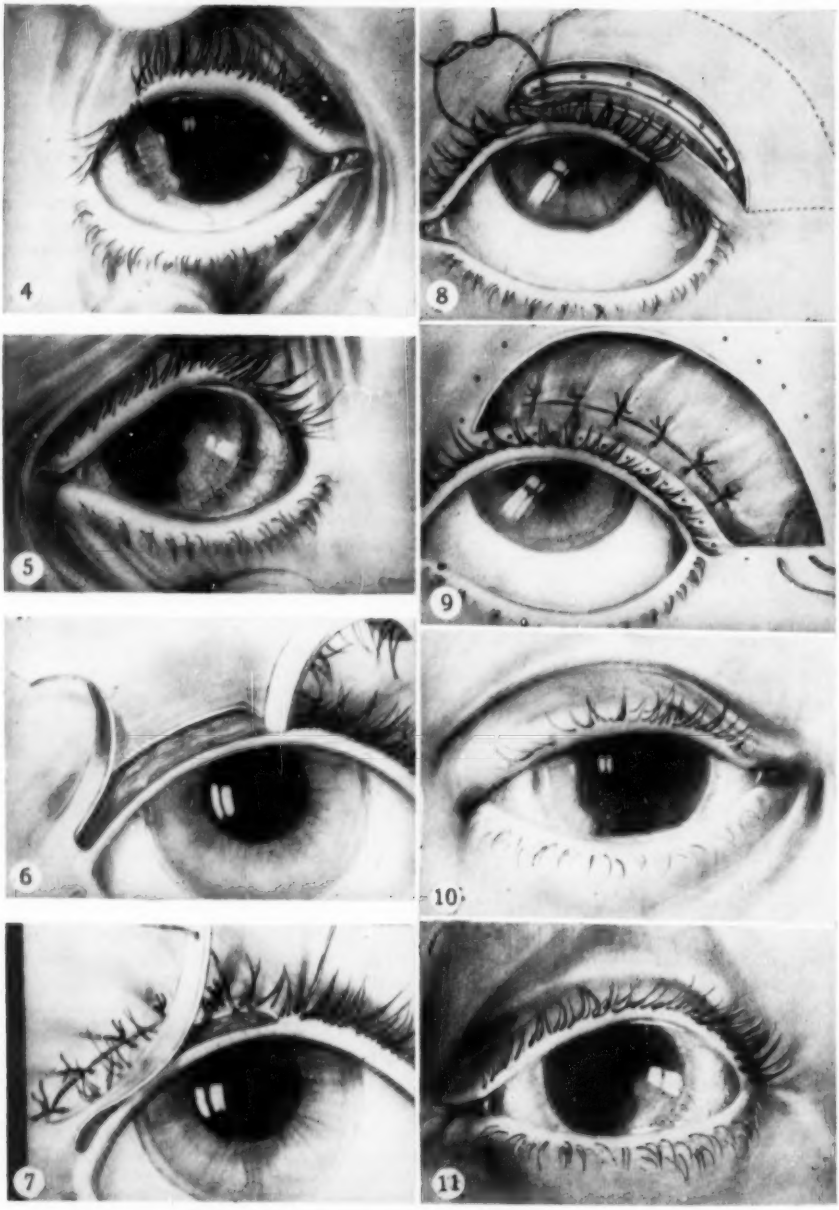
There was no aqueous flare, nor were there any free cells or synechiae. Muscle balance, fundi, and ocular tension were normal. Visual acuity was 20/40 in the right eye and 20/70 — 2 in the left, without correction. On refraction, it was 20/30 in the right eye with +1.50 sph. ⌒ —0.50 cyl. × 90, and 20/70 +2 in the left eye with +1.00 sph. ⌒ —5.00 cyl. × 80. It was felt that the reduction in visual acuity was principally due to a band of corneal haze produced by the advancing edge of the keratitis.

In June, 1953, operation was done to save the cornea from further involvement. The choice of operations available was great, since over 100 procedures have been developed for treatment of similar entropion with trichiasis caused by trachoma. Since the trichiasis in the right eye was so well localized laterally, it was felt that only the trichiasis, and not the entropion, required treatment. Consequently, a transposition of flaps was performed. The lower portion of the lateral half of the lids was formed into two horizontal tongues, one of which bore lashes, as shown in Figure 6. These tongues were then transposed and sutured into position with 6-0 black nonabsorbable surgical (silk) interrupted sutures, as shown in Figure 7.

Because greater corneal damage was present in the left eye, a more extensive procedure was indicated. Therefore, a slightly modified Streatfeild-Snellen procedure was done. A horizontal incision was made through to the tarsus about 3.5 mm. from the lid margin; the soft tissues were retracted, and a horizontal wedge of tarsus, apex posterior, and about 20 mm. long, was removed. The tarsal plate felt completely normal except for its accentuated concavity; there was no hypertrophy, irregularity, or atrophy. The cut surface of the slim wedge removed appeared to be normal. The skin was undermined extensively superiorly and laterally, as shown in Figure 8; then a D-shaped section of skin was removed. This was done, since it was felt that relatively taut skin would help splint the tarsal segments in proper position until they were united. The tarsal trough was closed with a line of interrupted 6-0 medium chromic surgical sutures, as shown in Figure 9. The orbicularis was then closed with a few inverted sutures of the same material. The skin was closed with interrupted 6-0 black silk sutures.

The eyes were closed with two double-armed 4-0 black silk marginal sutures tied over rubber pegs, and Wheeler compression dressings were applied. These were left undisturbed for five days. On the sixth day the sutures were removed. At this time, the tongues in the right lid lost their good apposition, causing some thickening of the lid margin postoperatively, as seen in Figure 10.

Six months later the patient had the appearance shown in Figures 10 and 11. The margin of the right lid remained somewhat thickened, and some of the cilia which had fallen out had not grown back. The left side was quite satisfactory except for a slight tenting just lateral



Figs. 4-11.—*4*, right eye—artist's preoperative sketch.

5, left eye—artist's preoperative sketch.

6, right eye—artist's operative sketch. Stage I: The flaps are dissected out.

7, right eye—artist's operative sketch. Stage II: The flaps have been transposed and sutured into position.

8, left eye—artist's operative sketch. Stage I.: A wedge of tarsus has been excised and the skin undermined.

9, left eye—artist's operative sketch. Stage II: The tarsus has been closed and a D-shaped section of skin removed.

10, right eye—artist's postoperative drawing.

11, left eye—artist's postoperative drawing.

to the midline, as seen in Figure 11. Figure 12 shows how the patient could comfortably open her eyes widely at that time. Biomicroscopy showed far fewer dilated, angry vessels, and the haze of advancing keratitis had disappeared. With correction similar to that given before, she could now see 20/20 with the right eye and 20/30 with the left. The high astigmatism against the rule, presumably from her corneal scarring, remained unchanged.

The exact nature of the condition remains somewhat obscure. We believe that the report of its having been present since birth was reliable, especially in the face of the other congenital anomalies in the sibship. The appearance suggested that there might have been an inflammation of the conjunctiva, but not of the tarsus. Theoretically, this could have occurred just prenatally, between the time the eyelids open and term, the irritant being an amniotic toxin. Certainly none of the reasons given by other authors would apply here. Himly [10] long ago stated that in late fetal life the edges of the lids tend to turn markedly inward and that this condition could persist, causing entropion. However, he also stated that real congenital entropion never occurs. The cases of Harlan [4] and de Schweinitz † were attributed to deficiency of the tarsal plates and were marked by rents in the conjunctiva and lack of adhesion

Fig. 12.—From a Kodachrome, showing how freely the eyes could be opened six months after operation.

between the latter and the tarsus. Redslob [8] believed that the entropion in his case was due to the thickening of the tarsus caused by the double row of Meibomian glands. Arkin [7] considered the entropion in his case to be due to excess of skin. Writers on entropion of the lower lids have variously ascribed the condition to hypertrophy of the marginal or palpebral portion of the orbicularis, or to failure of support in cases of microphthalmos. It is unlikely that we shall ever have a series large enough to ascertain definitely the etiology of this condition.

SUMMARY

A unique case of congenital entropion of the upper lids, accompanied by tetrastichiasis, trichiasis, palpebral hyperpigmentation, and mental deficiency, is presented. The subject was a member of a sibship tainted with congenital anomalies. We assume that the condition was caused by a prenatal conjunctival irritation, occurring late in development. Progressive superficial keratitis induced by the trichiasis was threatening vision. This process was satisfactorily stayed by plastic surgery.

† de Schweinitz, G., in discussion on Harlan.[4]

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CORNEAL TRANSPLANTATION

A Review of 365 Operations

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THE WAVE of enthusiasm over the promise of keratoplasty during the 1940's, particularly in the United States, raised many exaggerated hopes. Excessive publicity attendant on the establishment of eye banks misled both the public and some members of the medical profession as to the potentialities of the corneal graft technique.* The need was apparent for careful evaluation of large series of corneal graft cases to answer these specific questions: When should a corneal graft be done? What is the criterion of a successful operation? What is the probability of success in unselected series of cases? In selected cases?

Elschnig,[6] Filatov,[7] Thomas,[10] and Castroviejo † had provided some partial answers. Their work aside, few surgeons had had experience with more than a very small series of cases, a lack which posed a major difficulty for a comprehensive evaluation of results. Owens and his collaborators [9] met this problem by pooling the experience of many surgeons at clinics in neighboring cities. In their 1948 report on the series of 417 cases, the authors demonstrated sharp differences in operative clarity in various diagnostic groups. Later, Stansbury,[10] in a composite survey of 165 grafts performed by several surgeons, disputed the correlation between the original cause of corneal disease and the graft success, which he equates

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* References 12 and 15.

† References 1 through 3.

with visual improvement. The results of 365 corneal grafts performed at the Manhattan Eye, Ear and Throat Hospital, all but 18 of them by me, are analyzed in this paper for further light on the results of keratoplasty.

PROGNOSIS AND GRAFT CLARITY

Everyone agrees that the final results hinges largely on case selection. While the criteria of selection are in dispute, broad outlines of agreement among the various studies emerge. Repeatedly reports have stressed the inverse correlation between the size of the corneal opacity and the final result,‡ the presence of some healthy corneal tissue of the host being considered vital to the transparency of the graft.§ Filatov reported in 1935 on 96 penetrating grafts, 14 of which remained clear. In each successful case some transparent corneal tissue was present preoperatively. Castroviejo,[1] in 1938, in a summary of more than 100 cases, 40% of which had a successful result, comments that a transplant seldom remains transparent if it is entirely surrounded by scar tissue. A negative correlation is also reported between graft clarity and preoperative vascularization.‡

Criteria of Castroviejo.—The significance of preoperative vascularity and the size of the corneal opacity is implicit in the prognostic criteria devised by Castroviejo,[3] in 1946. For example, he puts a condition of central corneal opacity and mild interstitial keratitis in the presence of healthy corneal tissue in the very favorable category, together with keratoconus, which is commonly accepted as the most promising type of corneal opacity. On the other hand, extensive interstitial keratitis is held less favorable for operation, as is extensive corneal opacity without vascularization. Both extensive opacity with vascularized pannus and scarring which includes the limbal and pupillary areas are rated as unfavorable for keratoplasty.

Subsequently, several authors have analyzed the results of corneal graft series in terms of Castroviejo's categories and have reported substantial correlation between prognosis and final clarity.

Owens and his co-workers [9] affirmed the importance of case selection for operative success. They found, too, that the larger the corneal opacity and the greater the extent of vascularization, the less the chance of obtaining a clear graft. The ratio of transparent grafts in their entire unselected series of 417 cases was about 1 : 3. A breakdown of these cases into the categories outlined by Castroviejo showed the following pattern: Two-thirds of the grafts in the most favorable group, one-half of those in the less favorable group, and only one in eight in the unfavorable and contraindicated group remained clear. In agreement with Castroviejo's criteria, the best results were in cases of keratoconus, 65.2% of the grafts remaining clear. Next in order were the hereditary dystrophies, 58.8% of the grafts being transparent. These forms Castroviejo places in the less favorable column. Somewhat less than half the grafts done for interstitial keratitis and nonspecific scarring were successful. Poorer results were obtained in active keratitis or ulcer, scars from chemical burns, trauma, and gonococcal ulcer. No graft was successful in Fuchs's dystrophy.

Franceschetti and Bischler,[8] reporting on the results in 129 eyes, found a definite correlation between the original diagnosis and the graft transparency. They

‡ References 9, 13, and 16.

§ References 1, 7, and 16.

term the prognosis favorable in most, if not all, corneal dystrophies, in phlyctenular keratitis, and in corneal opacities caused by congenital or infantile glaucoma. Most unfavorable, in their opinion, are cases of severe chemical burn, severe corneal injury, or corneal degeneration in the aphakic eye.

A correlation between the cause of the corneal disease and the graft clarity was also found by Roberts [13] in his analysis in 1950 of 100 cases operated on by me at Manhattan Eye, Ear and Throat Hospital. His analysis also showed a correlation, as did Owens' series, between prognosis and result when these 100 cases were grouped according to the criteria of Castroviejo.

A similar correlation was found by Scheie,[14] who reported a series of grafts in 22 eyes. He added that, while it is advisable to operate under the favorable conditions outlined in the first two categories of Castroviejo, a transplant is also justified in less promising cases if the patient has nothing to lose and may benefit. Hesitation is indicated, however, in cases of unilateral involvement and in one-eyed persons.

Significance of Preoperative Vessels and Opacity Size.—Stansbury, however, in his analysis of 165 corneal grafts done by several surgeons, found only a slight relation between preoperative diagnosis and the success of the graft, which he equates with visual improvement, and no correlation between prognosis and the result.[16] The incidence of successful operations in his series was fairly constant in the various diagnostic groups, with the exception of keratoconus, which gave the best result, and the group of chemical burn cases, all of which were failures. Stansbury agrees with the findings of Owens and other authors as to the inverse relation between graft clarity and preoperative vascularization and the extent of opacity. He goes further, stating that only these two factors, plus the presence or absence of other pathological conditions of the eye, showed a definite correlation with the final visual result. The prognosis in each case should depend on the presence or absence of these pathological features, and should be individual.

Stansbury, however, qualifies the relation between opacity size and result. Nearly as good visual results were secured in opacities covering more than one-half of the cornea as in those less than one-half in size, while no grafts done in opacities extending over the whole cornea were successful. He suggests that the size of the opacity is not important provided that the graft postoperatively will be surrounded, or nearly so, by clear corneal tissue, but he does not state the amount of clear cornea.

Moreover, the visual results in eyes with little corneal vascularization equaled those in cases with no preoperative vessels. While the number of cases classified as having a little corneal vascularization was small, Stansbury offers the possibility that slight preoperative vascularity may actually be a favorable factor in corneal transplantation. (This has not been my experience.)

My findings in the current series of 365 operations are in agreement with these writers as to the significance of preoperative vascularization and the size of the opacity for the final result. Experience with this series would also seem to bear out Stansbury's view regarding the lack of correlation in many cases between the original diagnosis and the graft result. All corneal graft cases do not lend themselves to exact prognostic classification. In every case of scarring, whether caused by a stab wound, other trauma, a chemical burn, trachoma, interstitial keratitis, or

Gonococcus ulcer, operative success depends on the degree of scarring, the amount of vascularization present, and the time interval following subsidence of the active inflammatory process.

I question, however, the categories used by Stansbury,[16] Owens,[9] and Roberts [13] in evaluating the degree of preoperative vascularization. These categories were as follows: none, slight or little, moderate, and extensive. In my opinion, there is no way of tabulating the judgments of individual surgeons. Moreover, a preoperative evaluation can in any case be only relative. A finding of slight vascularization might be made in the presence of a single vessel; yet this one vessel could have a very adverse effect on the graft. In speaking of extensive vascularization, one should indicate, whenever possible, whether the vascularization is superficial or deep. Extensive vascularization from the conjunctival vessels is less detrimental to graft transparency than are the deep vessels, some of which arise from branches of the ciliary vessels. The presence of a large deep vessel is usually a contraindication to operation unless it can be destroyed by beta radiation therapy before the graft is done. This treatment is given preoperatively in nearly all my cases when vascularization is present. It must be borne in mind, however, that clear grafts may be obtained in cases in which not all vessels have been destroyed by preliminary beta radiation. This therapy may, in some instances, inhibit the growth of vessels without actually causing their destruction.

Criteria of Operative Success.—Stansbury differs sharply with the common acceptance of graft clarity as the criterion of success in keratoplasty.‖ He states that a clear graft does not necessarily mean a better eye. The only logical index of success, in his view, is visual improvement to better than 20/200, the threshold of industrial blindness. From an ideal standpoint, one must agree with Stansbury that visual improvement is the test of keratoplasty. Yet, as Thomas [19] pointed out in 1937, a transparent graft means a successful operation, even though other ocular defects prevent visual improvement. In the view of Owens and associates,[19] the criterion of visual gain alone would necessitate exclusion of clear grafts in which vision is limited by such conditions as cataract. In the present series of cases, the measure of operative success was absolute graft clarity, or, in a very few cases, slight haziness combined with great visual improvement.

OPERATIVE TECHNIQUE

My series includes a total of 365 corneal grafts performed at the Manhattan Eye, Ear and Throat Hospital, all but 18 of them by me. Of these grafts, 311 were penetrating; 54 were lamellar. Much valuable knowledge has been gained from personal experience with this operative series. While it is not the purpose of this paper to discuss the various techniques used in keratoplasty, a brief outline of the operative technique employed is presented in the following paragraphs, together with illustrative drawings (Figs. 1 to 5), in order that others may benefit from this experience.

A round graft was used in every case but one, in which a square button was employed. The postoperative care given these patients was similar to that in cataract operations. Sutures are usually taken out beween the 10th and the 14th postoperative day.

Much controversy still exists over certain phases of the operation. The accompanying illustrations (Figs. 1 to 5) demonstrate a standard technique, employed in all except a few instances in which direct sutures were used. It is my opinion that direct sutures should always be used

‖ References 15 and 16.

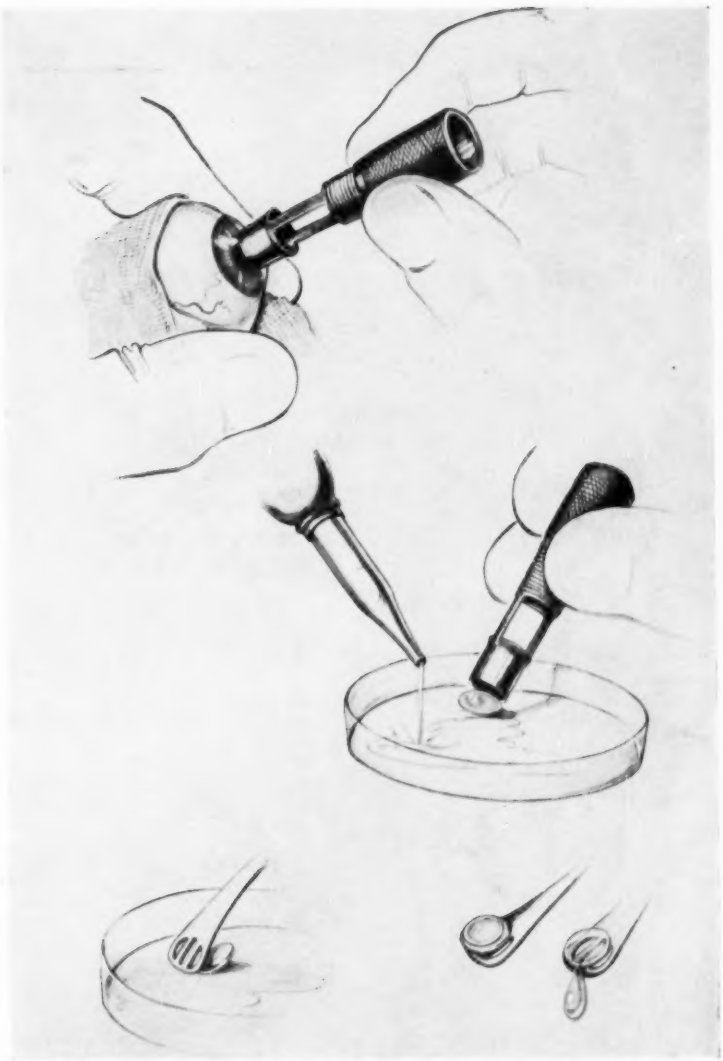

Fig. 1.—Cutting of donor window; placing of graft in Petri dish with endothelial surface uppermost. Saline drop must be drained off in order to be certain concave surface is uppermost.

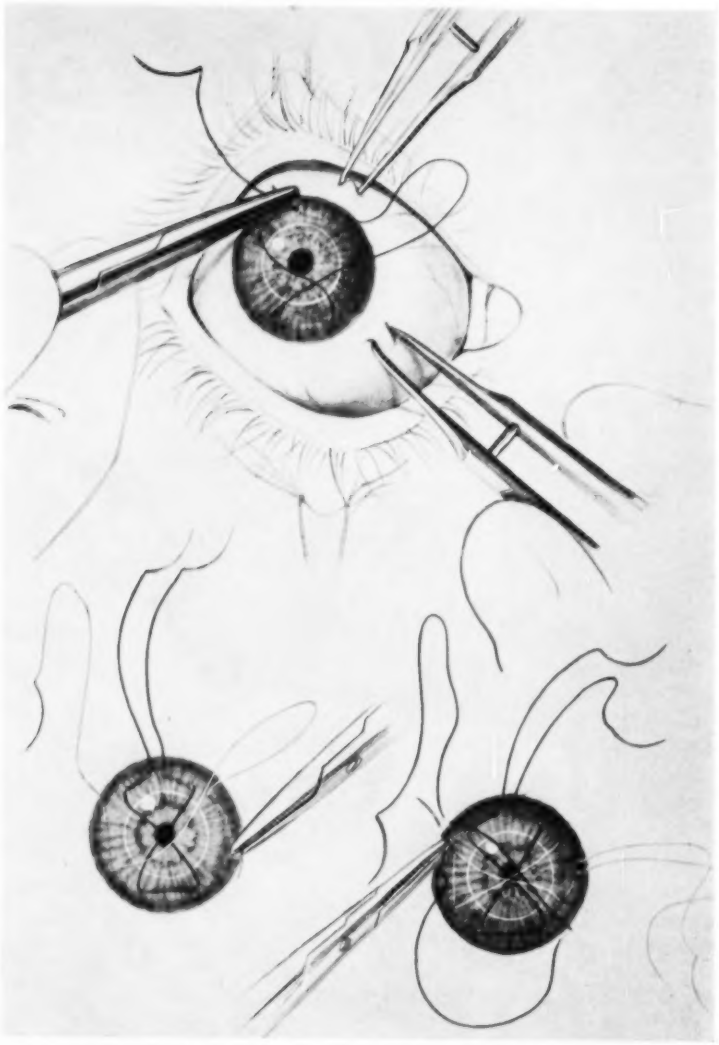

Fig. 2.—Outline has been made with trephine and accentuated by means of fluorescein. Note that the pupil has been contracted before operation with a miotic. Preplaced 000000 sutures are employed—two figure-of-eight, one white and one black, and one parallel suture from limbus to limbus.

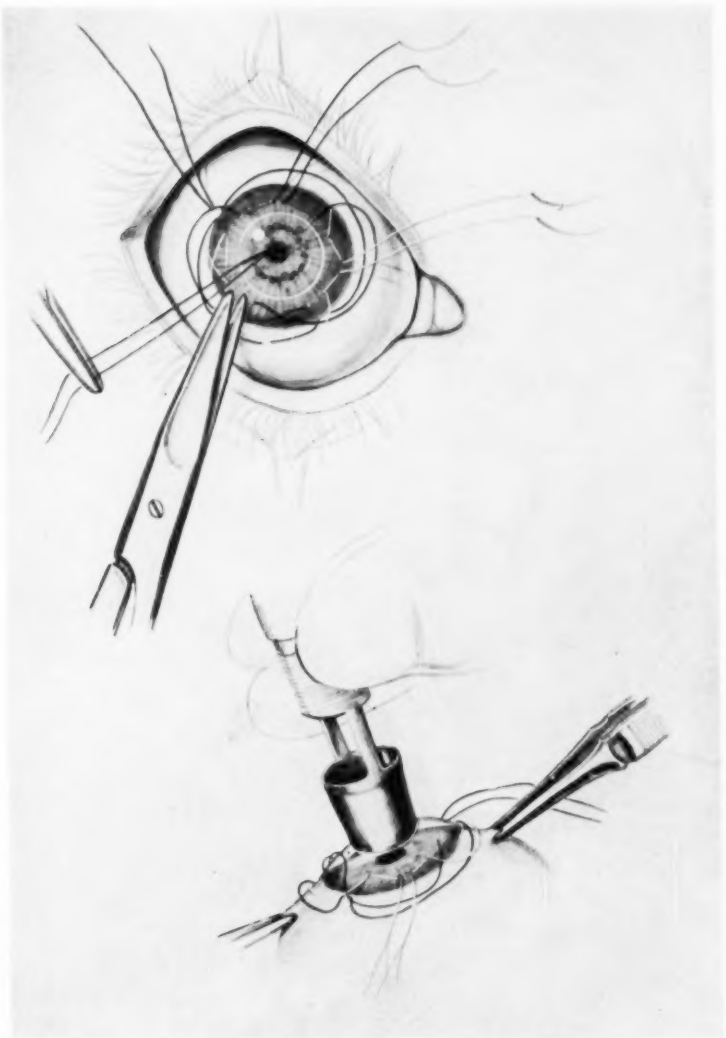

Fig. 3.—Central traction suture tied and cut. Anterior chamber perforated with trephine.

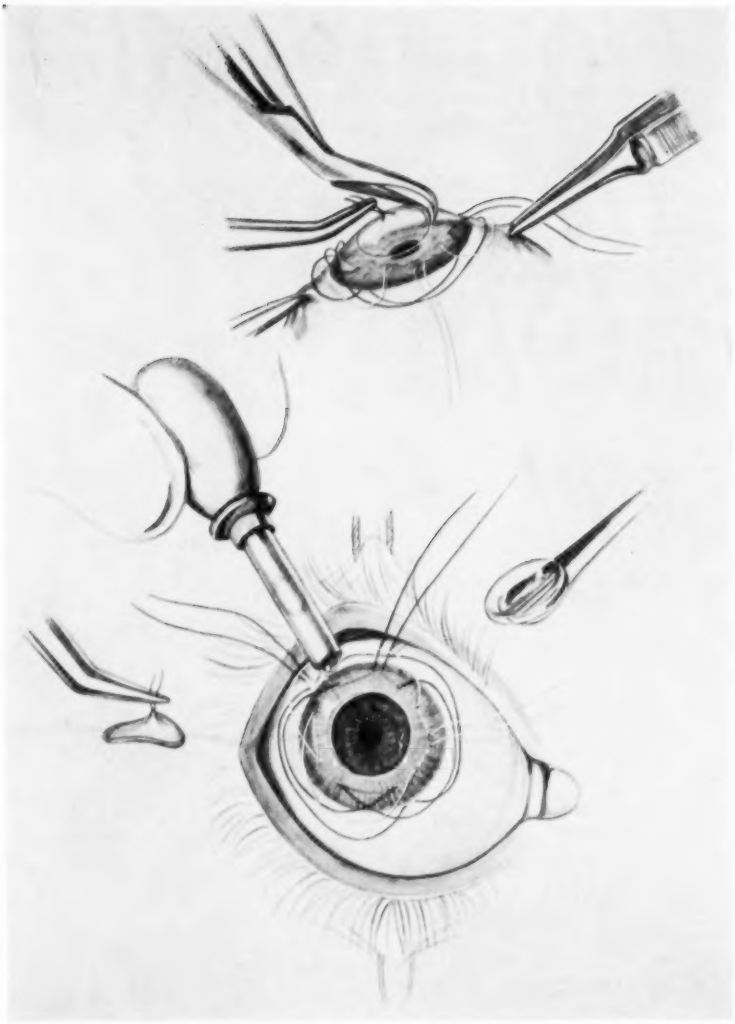

Fig. 4.—Button cut out and the eye flushed out with saline.

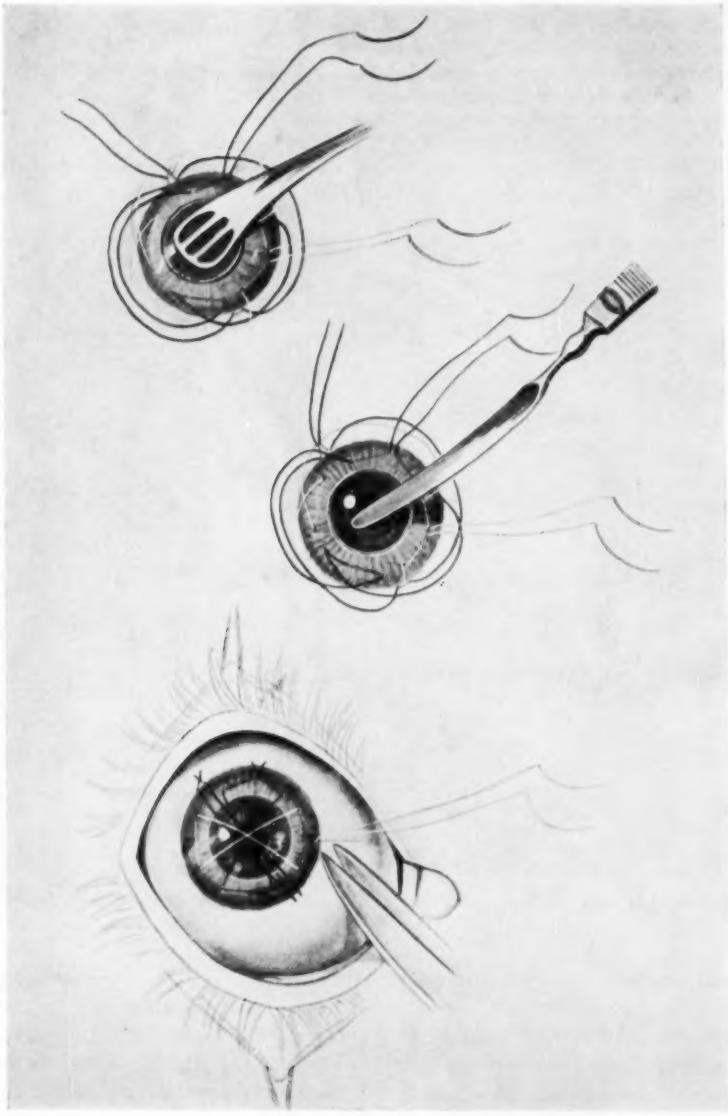

Fig. 5.—Graft in position and molded in place by means of flat surface of spatula. Sutures tied; ends cut. Cataract dressing is then applied to both eyes.

in grafts over 6 mm. in size. In this whole series of cases, overlying sutures were employed in grafts 6 mm. or less in size, while in those of more than 6 mm. direct sutures were used.

The technique for lamellar transplantation is very similar to that used in penetrating grafts. The guard on the trephine is set to cut to a depth of only 0.5 mm. The dissection of the lamellar graft is done using a Bard-Parker No. 15 blade.

Preoperative and postoperative pictures are available on nearly all the cases reported. Figures 6 to 11 illustrate cases of unusual interest.

ANALYSIS OF RESULTS IN 365 CORNEAL GRAFTS

The data for the analysis were drawn from the careful records maintained on each patient. These include the clinic card, the hospital record, the office record in

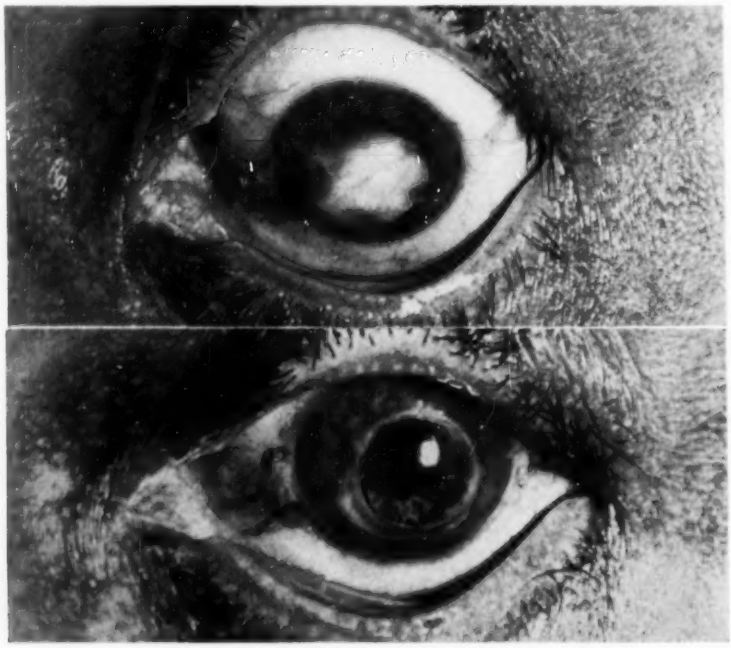

Fig. 6 (Case 37, Table 1).—Conical cornea with large cystic scar: Upper, preoperative; lower, postoperative.

private cases, and a special record card used in the corneal clinic (Fig. 12) which furnishes an up-to-date summary of the important facts in each case. As the physician makes his first examination of a patient in the corneal clinic, he dictates the preoperative diagnosis and a description of the corneal scar to a specially trained secretary. This information, together with a description of the operation and the postoperative course, is entered on the special record card. A careful follow-up is maintained on nearly all patients. Information on patients living at great distances is secured from their local physicians.

A follow-up period of two months was taken as the minimum in computing the operative results. In the majority of cases, however, the patients had been followed for a much longer period.

Graft Clarity.—The 365 operations in the series were performed on 330 eyes, two successive grafts having been done on the same eye in 25 instances and three grafts in 5 instances. In the computation of operative results, the clarity of the last graft only was counted in cases of multiple operations on the same eye, except in a special tabulation of lamellar grafts.

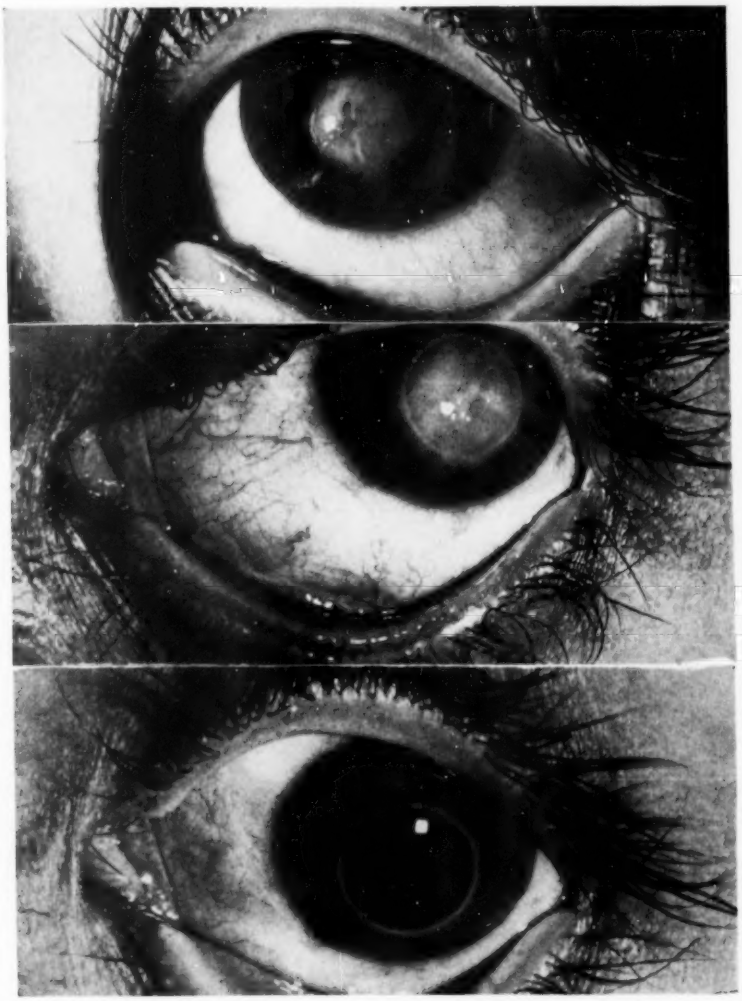

Fig. 7 (Case 27, Table 1).—Upper, conical cornea with large cystic scar; middle, first operation a failure; lower, after second operation (transplant of same size as the first).

In 13 cases the graft was clear on discharge, but there was no follow-up. Eighteen grafts were followed for less than two months. Of these, 10, or 55.6%, remained clear during the period of observation; 8, or 44.4%, clouded.

The remaining 299 grafts were followed for two months or longer. Of these, 193, or 64.5%, remained clear; 106, or 35.5%, clouded. If the 12 grafts done for therapeutic purposes which clouded are excluded from the computation, this percentage of clear grafts rises to 67.2%. In the unselected series, therefore, the patient had approximately two chances in three of obtaining a clear graft.

Roberts [13] reports an incidence of 55% clarity for the 100 grafts reported in his series. Franceschetti and Bischler,[8] in their analysis of grafts in 129 eyes, set the chances of postoperative clarity at about 50%, provided that cases of severe chemical

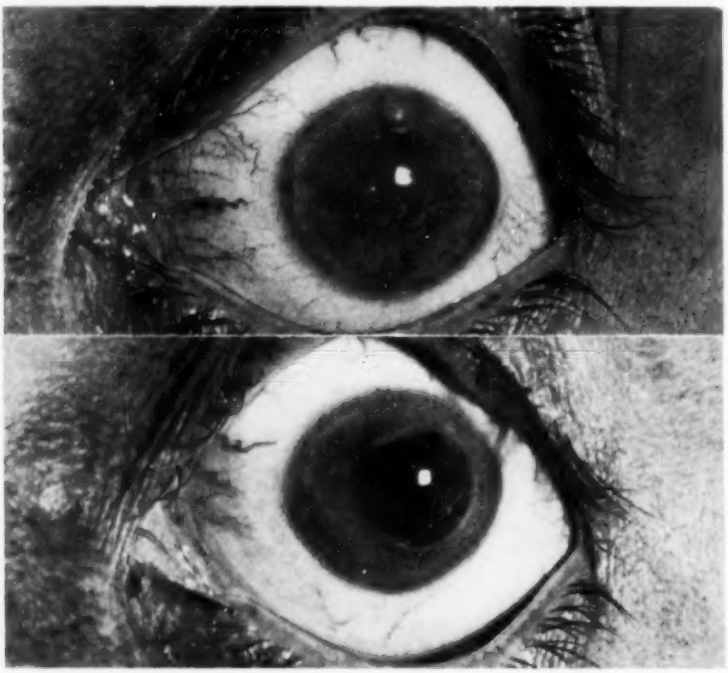

Fig. 8 (Case 3, Table 3).—Groenouw's dystrophy: Above, preoperative; below, postoperative.

burn, other severe corneal injuries, and corneal degeneration in the aphakic eye are excluded. In the study of Owens and associates,[9] 36.5% of grafts followed four months or longer remained clear, or better than 1 in 3. Stansbury,[15] who evaluated his series of 165 grafts in terms of visual improvement to better than 20/200, reports such gain after 22 operations, or 13% of the series. Clear grafts occurred twice as often—in 26.7%.

A word of interpretation as to the role of case selection in the result is pertinent here. The relatively high percentage of clear grafts—64.5%—among my cases resulted in part from the presence in the series of a large number of keratoconus cases, commonly considered very favorable for keratoplasty. Of the 193 clear grafts followed for two months or longer, 75, or 38.9%, had been done for keratoconus.

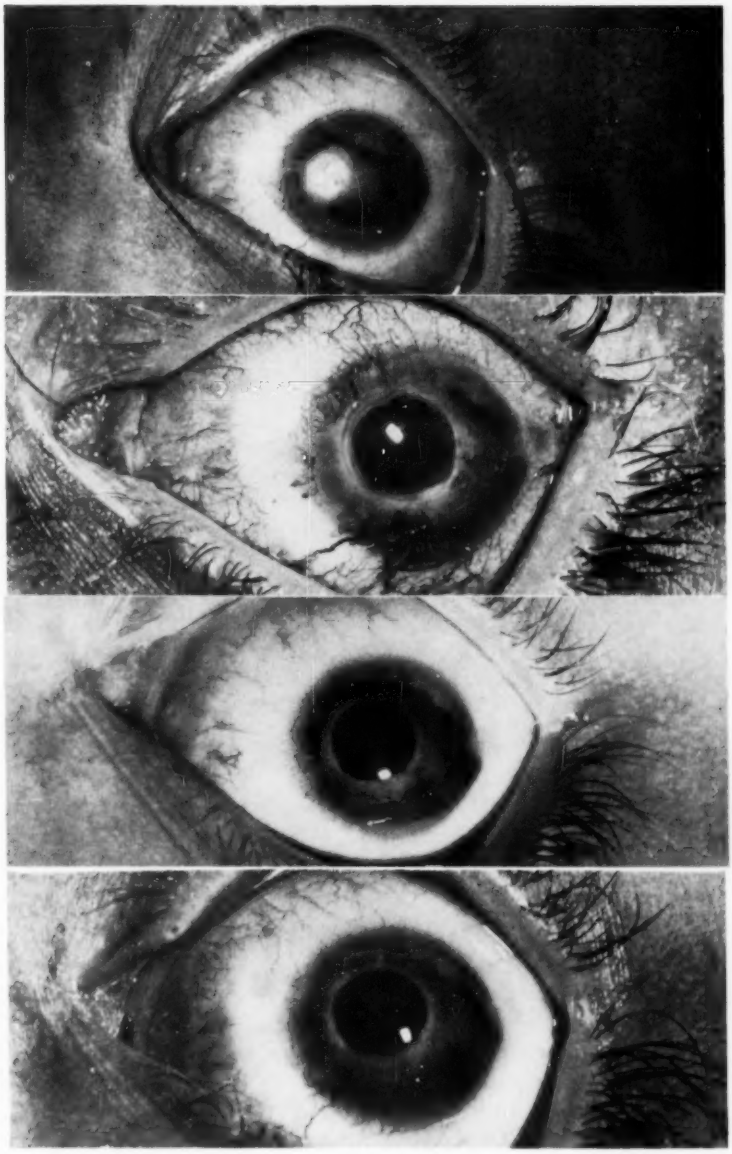

Fig. 9 (Case 1, Table 4).—Adherent leucoma with optical iridectomy: Top, preoperative; next below, one month after operation; next below, two months after operation; bottom, two years after operation. This case has previously been reported.

The 365 operations in the current series are summarized in Tables 1 to 10. The results according to diagnostic groups in cases followed two months or longer are set forth in Table 11, together with the findings in other reported series.

Keratoconus.—One point of concurrence in the literature is the excellent outlook for a clear graft in the patient with conical cornea. Castroviejo [3] puts this diagnosis in the very favorable category in his classification scheme and, in a later paper,[4]

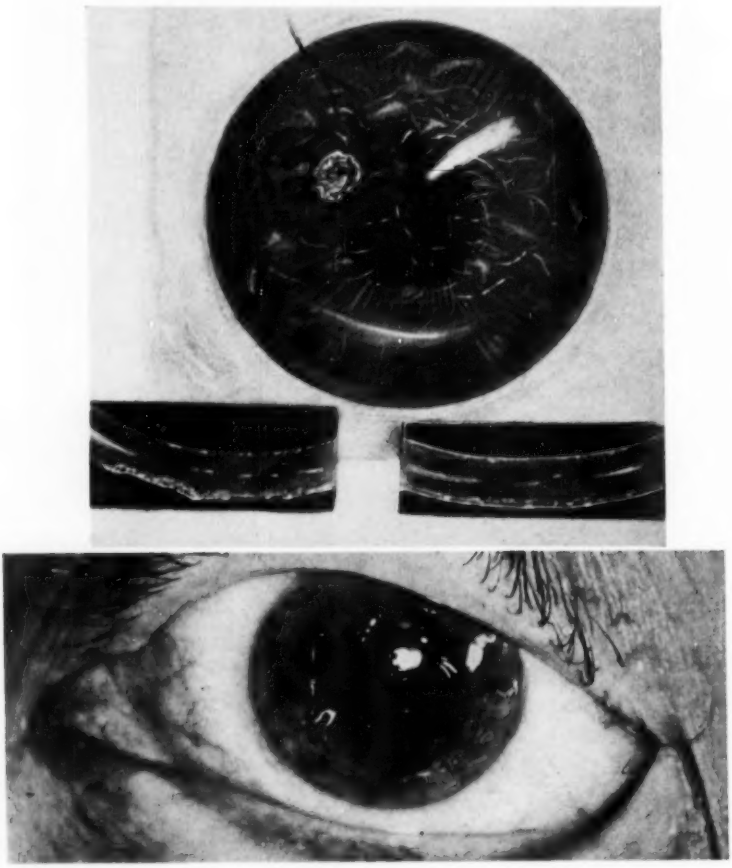

Fig. 10 (Case 5, Table 5).—Upper, interstitial keratitis with scar of old ulcer; lower, after third operation.

states that the keratoconus patient has a 75 to 90% chance of visual improvement after keratoplasty. In Roberts' series,[13] 85% of the 20 patients with conical cornea had clear grafts, while Owens and associates [9] report a transparent result in 65.2% of 63 cases. Keratoconus was one of the two diagnoses that Stansbury found showing a correlation with the operative result.[16] Ten of the 22 grafts in his series that resulted in visual improvement were done for conical cornea.[15]

Of the 330 eyes operated on in my series, 96 had a diagnosis of conical cornea (Table 1). Eighty-four of the grafts were followed for two months or longer, and of these, 75, or 89.3%, were clear. Four grafts had less than two months' follow-up, three of these being clear, while eight were transparent on discharge, with no follow-up. One enucleation was done four years postoperatively. The graft had remained clear for two years, then clouded in the presence of glaucoma, which apparently had existed preoperatively.

In the majority of these keratoconus cases, a button 6 mm. in diameter was used. The optimum graft size is a point at issue among eye surgeons. Stansbury [16]

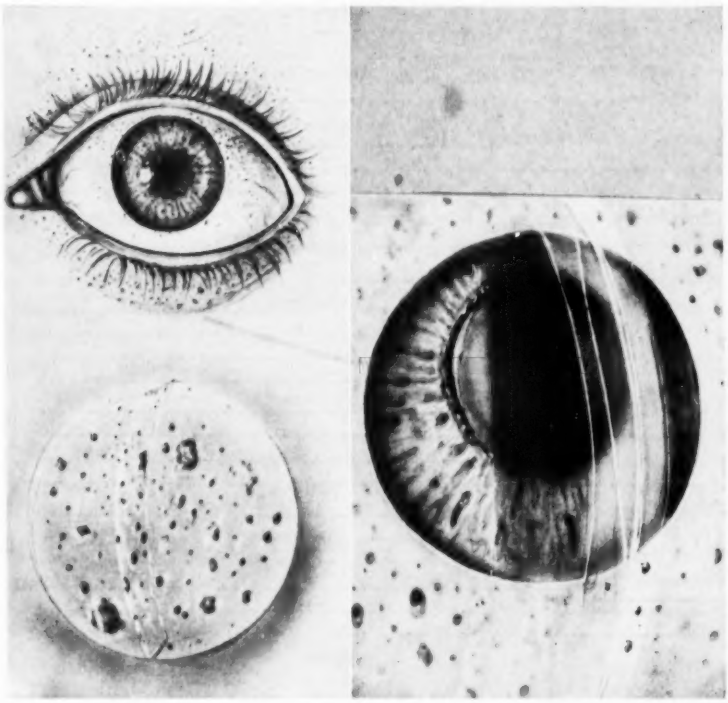

Fig. 11 (Case 15, Table 9).—Left, powder blast, with slit-lamp drawing; right, slit-lamp drawing after lamellar graft.

analyzed the results in his whole series in relation to graft size. Grafts 6 to 6.5 mm. in diameter gave a higher incidence of success than did smaller buttons. I feel, however, that with larger grafts, that is, those more than 6 or 7 mm. in diameter, there is likely to be an increase in postoperative complications.

Groenouw's Dystrophy.—The hereditary dystrophies are termed favorable for keratoplasty by Stansbury [16] and by Franceschetti and Bischler,[8] although Castroviejo [8] lists them in his less favorable category. Clarity was achieved in all the eight cases reported by Roberts,[13] while Owens and colleagues [9] found this group second in order of success after conical cornea, 58.8% of 17 grafts remaining transparent.

In my series, also, Groenouw's dystrophy came next to keratoconus (Table 3). Of 28 grafts followed two months or longer, 24, or 85.7%, were clear. Three grafts had less than two months' follow-up, two of these being transparent and one fairly clear.

MANHATTAN EYE, EAR AND THROAT HOSPITAL - CORNEAL GRAFT CLINIC

RECIPIENT EYE

Case #_____ Clinic #_____ Age____ Sex____ Color____

Name_____ Admission #_____

PRE-OP. EXAM. Date_____

Diagnosis_____

Corneal Opacity_____ Anterior Synechia_____

Vessels_____

Pre-Op. Vision: With glasses_____ Without glasses_____

Previous Operations_____

Pre-Op. Rx._____

OPERATION Date_____

Eye_____ Size of Graft_____ Technic_____

P. O. Course_____

Subsequent Course_____

Final Vision_____ Date_____

Final Graft Clarity_____ Date_____

Final Refractive Error_____ Date_____

Comments_____

DONOR EYE

Age_____ Sex_____ Color_____ Eye Bank #_____

Time between Death (or Enucleation) and Graft_____

Time in Bank_____

Clinical Pre-Enucleation Diagnosis_____

Final Histopathologic Diagnosis_____

Notes_____

CASE #_____ NAME_____ DOCTOR_____

Fig. 12.—Sample record card for corneal graft cases.

Nonspecific Scarring.—Third in percentage of clear grafts in my series was the general category of nonspecific scarring. This group ranked fourth in the series reported by Owens and associates,[9] with clear grafts in 46.9% of 141 cases, and fourth in Roberts' series, in which 42.8% of 42 transplants were transparent.[13]

Nineteen of my cases had this diagnosis (Table 8). Of 16 grafts followed two months or longer, 11, or 68.8%, were clear. Three were followed less than two months, all of these being cloudy.

Scarring from Inactive Ulcer.—Twenty cases in the current series had a diagnosis of scar from inactive ulcer. One graft was clear on discharge, with no follow-up, while one followed for less than two months was clear. Of 18 grafts followed two months or longer, 12, or 66.7%, were transparent and 6 cloudy (Table 7).

Interstitial Keratitis.—That a corneal opacity resulting from interstitial keratitis is well suited for keratoplasty, provided it is not too extensive, is generally agreed. Mild interstitial keratitis, in the presence of healthy corneal tissue, ranks with Castroviejo's very favorable diagnoses,[8] while more extensive interstitial keratitis is in his less favorable category. Stansbury [16] terms this condition promising for keratoplasty, although he points out that the results for this group in his series were not significantly better than the average. Elschnig,[6] in 1930, reported 17 clear grafts among 26 cases of interstitial keratitis, with marked visual improvement in all 17 cases. Approximately half the grafts done for syphilitic interstitial keratitis in the series of Owens and associates,[9] and 6 of 12 grafts done for this condition in Roberts' series,[13] remained clear.

In the current series, 45 grafts in cases of interstitial keratitis were followed for two months or longer (Table 5). Twenty-eight of these, or 62.2%, remained clear. One graft followed for less than two months was clear, while in two cases the grafts were clear on discharge, with no follow-up. Panophthalmitis developed in one eye, necessitating enucleation.

Leucoma.—Ten cases in my series had a preoperative diagnosis of leucoma (Table 4). One graft was clear on discharge, with no follow-up. Nine were followed for two months or longer, five of these, or 55.6%, remaining clear. There was one enucleation, resulting from panophthalmitis.

Active Ulcer.—Six therapeutic grafts were done in cases of active ulcer—four lamellar and two penetrating grafts (Table 7). The results were unexpectedly good, although the group is too small to constitute a statistical sample. Two grafts were clear; another was partly clear, with great improvement in vision. Three patients obtained relief of symptoms.

Owens and associates report clear grafts in 23.1% of 13 cases of active ulcer or keratitis, a finding which they consider surprisingly good.[9]

Trachomatous Scarring.—Eleven cases of trachomatous scarring were followed two months or longer. Five of these, or 45.5%, had clear grafts (Table 6).

Chemical Burns, Explosion Injuries, and Traumatic Wounds.—The generally negative view expressed in the literature regarding keratoplasty in the chemical or lime burn is substantiated in my experience. The results are nearly always poor. Elschnig [6] terms total scarring of the cornea following alkali burns most unfavorable for keratoplasty. Castroviejo [3] includes in his unfavorable category extensive corneal opacities with pannus-type vascularization, which, he writes, are generally caused by chemical, flame, or molten-metal burns. Of nine cases of chemical burn in Stansbury's series, none had a good result after keratoplasty.[15] Stansbury [16] lists the

TABLE 1.—*Results of Corneal Grafts in Keratoconus**

Case No.; Patient	Sex; Age., Yr.	Eye	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1 H. B.	M 30	R. E.	Cystic scar	Some hair-like	H. M. at 2 ft.	6 mm. Penetr.	2 mo. postop., w. −6.00 sph ⊃ −3.00 cyl, ax 40 = 20/200	Clear
		L. E.	—	—	H. M.	6 mm. Penetr. 2 mo. after operation	6 mo. postop., w. −2.00 sph ⊃ −4.00 cyl, ax 80 = 20/50	Clear
2 M. C.	M 22	R. E.	Dense, central	—	20/70 w. cont. gl.	6 mm. Penetr.	2 mo. postop., w. −6.00 sph = 20/20−2	Clear
3 R. B.	F 53	R. E.	—	—	3/200	6 mm. Penetr.	7 mo. postop., w. +5.00 sph ⊃ −7.50 cyl, 30 = 20/30	Clear
		L. E.	Small cystic scar at apex	—	20/100 without cor.	6 mm. Penetr. 11 mo. after op. on R. E.	4 mo. postop., w. −2.00 sph ⊃ +7.00 cyl, ax 145 = 20/50	Clear
4 C. C.	F 24	R. E.	Central, slight	—	20/20+2 w. cont. gl.	6 mm. Penetr.	8 mo. postop., w. cont. gl. = 20/20	Clear
		L. E.	—	—	20/40 w. cont. gl.	6 mm. Penetr. 11 mo. after op. on R. E.	7 mo. postop., w. −3.00 sph ⊃ −3.00 cyl, ax 150 = 20/25	Clear
5 A. C.	F 32	L. E.	Central, at cone	—	20/200+1 without cor.; no improvement w. cor.	6 mm. Penetr.	3 yr. postop., 20/200; no improvement w. gl.	Clear graft; eye amblyopic
6 W. C.	M 17	L. E.	Very dense, central	Slight	L. P.	5 mm. Penetr.	2¼ yr. postop., 2/200	Clear graft; eye amblyopic
7 B. F.	M 33	L. E.	Dense	—	5/200	4.5 mm. Penetr.	1½ yr. postop., L. P.	Clear graft; poor vision due to glaucoma
8 M. F.	F 28	L. E.	Central, bullous	—	H. M.	6 mm. Penetr.	1½ yr. postop., w. cont. gl. = 20/20	Clear
		R. E.	Central, bullous	—	20/200 w. cor.	6 mm. Penetr. 10 mo. after op., L. E.	10 mo. postop., w. cont. gl. = 20/20	Clear
9 G. G.	F 38	R. E.	—	—	2/200 without cor.; w. cont. gl. = 20/50−1	6 mm. Penetr.	9 mo. postop., C. F.	Cloudy graft; ant. synechias present; synechiotomy unsuccessful
		R. E.	Cloudy graft; ant. synechias present	Some; destroyed by beta ray	1/200	6 mm. Penetr., 1 yr. 8 mo. after 1st op.	6 mo. postop., w. −5.00 sph = 20/100	Later lower part of graft clouded; ant. synechias developed
		R. E.	Persistent fistula of graft	—	2/200	6 mm. Penetr., 2 yr. after 2d op.	3 mo. postop., 4/200	Clear graft; cataract and anterior synechias present
10 M. H.	F 57	R. E.	Nebulous	—	H. M. at 2 ft. without cor.; 2/400 w. cor.	6 mm. Penetr.	2 yr. postop., w. −4.00 sph ⊃ −2.00 cyl, 180 = 20/50	Clear

11 F. H.	M 46	L. E.	Central	—	4/200	5 mm. Penetr.	2 yr. postop., w. cont. gl. = 20/20—2	Clear
12 E. B. J.	M 52	L. E.	Scar at apex; extreme conical cornea	—	H. M. at 2 ft.	6 mm. Penetr.	8 mo. postop., L. P.	Cloudy graft; immature cataract present; eye amblyopic (nystagmus and coloboma of iris present preoperatively)
13 B. K.	M 15	R. E.	Central ⅓ stroma	—	10/200 without cor.	5 mm. Penetr.	6 mo. postop., w. —2.50 sph. = 20/100	Clear in 4 quadrants; opacity arranged like cross
		R. E.	Moderate	—	10/200 without cor.; 20/200 w. cor.	6 mm. Penetr., 1 yr. after 1st op.	2 yr. postop., 20/30 without cor.	Clear
		L. E.	Central	—	20/200 without cor.	6 mm. Penetr., 1 yr. after 2d op. on R. E.	10 mo. postop., 20/40 without cor.	Clear
14 L. H. K.	F 64	L. E.	Dense, central	—	20/100 w. cor.	5 mm. Penetr.	2 mo. postop., w. —3.00 sph = 20/70	Clear
15 A. B.	F 45	L. E.	Very dense, central	—	20/100 without cor.	5 mm. Penetr.	4 mo. postop., w. —2.00 sph ⊃ +3.00 cyl, ax 80 = 20/100	Graft clouded later
		L. E.	Very dense, central	—	1/200	6 mm. Penetr., 1¾ yr. after 1st op.	2½ yr. postop., 20/25+3 without cor.; no improvement w. cor.	Clear
16 E. L.	M 38	R. E.	Central	—	2/300 without cor.	6 mm. Penetr.	3 yr. postop., 20/20 w. cor.	Clear
		L. E.	Central	—	20/100 w. cont. gl.	6 mm. Penetr., 3 yr. after op. on R. E.	No follow-up	Clear; no follow-up
17 L. L.	F 26	R. E.	—	—	2/200 without cor.	7 mm. Penetr.	2 wk. postop., w. +2.00 sph = 20/40	Clear, 9 mo. postop.
18 H. L.	M 40	L. E.	Nebulous	—	4/200 without cor.	6 mm. Penetr.	6 mo. postop., w. —3.00 sph ⊃ +5.50 cyl, ax 52 = 20/40	Clear
19 D. M.	M 18	R. E.	Central, stellate-shaped	—	20/100 w. cont. gl.	6 mm. Penetr.	5 mo. postop., w. —14.00 sph ⊃ —6.00 cyl, ax 160 = 20/40	Clear
20 E. M.	F 36	L. E.	—	—	3/200 without cor.	6 mm. Penetr.	2½ yr. postop., w. —4.00 cyl, ax 10 = 20/30	Clear
21 A. M.	M 36	L. E.	Central	—	5/200 w. cor.	6 mm. Penetr.	3 mo. postop., no improvement	Infection developed; graft clouded
22 Am. M.	M 23	R. E.	At apex	—	3/200 without cor.; 20/200 w. cor.	6 mm. Penetr.	6 mo. postop., w. —4.75 sph ⊃ —3.25 cyl, 20 = 20/25+4	Clear
23 W. P.	M 20	R. E.	Central	—	1/200 without cor.	10 mm. Penetr.	1¼ yr. postop., w. —10.00 sph ⊃ —1.50 cyl, ax 130 = 20/40+	Clearing
		L. E.	Central	—	10/200 without cor.	6 mm. Penetr., 1 yr. after op. on R. E.	3 mo. postop., w. —5.00 sph ⊃ —4.00 cyl, ax 90 = 20/40+1	Clear
24 A. R.	F 40	R. E.	Folding of Descemet's membrane	—	10/400 w. and without cor.	6 mm. Penetr.	2 yr. postop., w. —12.00 sph ⊃ — 3.00 cyl, ax 180 = 20/50	Clear
		L. E.	Central, faint	Peripheral, not marked	20/200 w. and without cor.	6 mm. Penetr., 7 mo. after op. on R. E.	1½ yr. postop., w. —9.00 sph ⊃ —3.00 cyl, ax 175 = 20/70+2	Clear

TABLE 1.—*Results of Corneal Grafts in Keratoconus**—Continued

Case No.; Patient	Sex; Age, Yr.	Eye	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
25 S. R.	F 26	L. E.	Nebulous	—	5/200 without cor.; 20/200 w. cor.	6 mm. Penetr.	10 mo. postop., w. −5.50 sph ⊃ −2.25 cyl, ax 100 = 20/30−2	Clear
		R. E.	Central, faint	—	3/200 w. and without cor.	6 mm. Penetr., 2 yr. after op. on L. E.	5 mo. postop., 20/200 without cor.; no improvement w. cor.	Infection developed; graft became opaque
		R. E.	Nearly opaque graft	Few, lower part	20/200 w. and without cor. 10 mo. after 1st op. R. E.	5 mm.	No follow-up	Graft clear on discharge
26 E. R.	F 48	R. E.	Central	—	4/200 w. and without cor.	4.5 mm. Penetr.	5 yr. postop., w. −8.00 sph ⊃ −1.00 cyl, ax 75 = 20/25+2	Clear
27 M. R.	F 21	L. E.	Bullous keratitis	—	20/200—1 w. and without cor.	6 mm. Penetr.	.....	Cloudy
		L. E.	Nebulous	One, small	2/200	6 mm. Penetr., 4 mo. after 1st op., L. E.	3 mo. postop., w. −3.00 sph ⊃ −2.00 cyl, ax 155 = 20/20−3	Clear; adhesions present below but did not interfere with transparency
28 I. R.	F 21	L. E.	Central	—	5/200 without cor.	6 mm. Penetr.	2½ yr. postop., w. −12.00 sph ⊃ +4.00 cyl, ax 125 = 20/30	Clear
		R. E.	—	—	20/200 without cor.	6 mm. Penetr., 2 yr. after op. on L. E.	1 yr. postop., w. −11.00 sph ⊃ +7.00 cyl, ax 125 = 20/70	Clear
29 P. S.	M 19	R. E.	Thinning, centrally	—	1/200 without cor.	6 mm. Penetr.	11 mo. postop., w. −7.50 sph ⊃ −5.50 cyl, ax 35 = 20/30−2	Clear
30 C. S.	M 42	R. E.	Fairly dense, central	—	1/200 without cor.	6 mm. Penetr.	2¾ yr. postop., w. −20.25 sph ⊃ −2.75 cyl, ax 180 = 20/50−3	Clear
31 H. S.	M 39	L. E.	—	—	20/50 w. cor.	6 mm. Penetr.	6 mo. postop., w. cont. gl. = 20/30	Slightly hazy
32 B. S.	M 25	R. E.	Moderate	—	20/200 w. cont. gl.	5 mm. Penetr.	2 mo. postop., w. cont. gl. = 20/20	Clear
		L. E.	Slight	—	1/200 without cor.; w. cont. gl. = 20/30	6 mm. Penetr., 2½ yr. after op. on R. E.	5 mo. postop., w. −4.00 sph = 20/20	Clear
33 B. Se.	M 29	L. E.	Central	—	4/200 without cor.; w. cont. gl. = 20/50	6 mm. Penetr.	1½ yr. postop., w. −2.00 sph ⊃ −10.00 cyl, ax 165 = 20/30	Clear
34 R. S.	M 29	R. E.	Central	—	2/200 without cor.	4.5 mm. Penetr.	.....	Graft clouded; donor eye believed poor
		R. E.	Moderate, pericentral	—	20/200+ w. ph	6.5 mm. Penetr. 1½ yr. after 1st op.	1½ yr. postop., w. cont. gl. = 20/20	Clear
35 R. Sm.	F 26	L. E.	Central fourth	—	10/200 w. cor.	4.5 mm. Penetr.	3 yr. postop., w. −13.00 sph ⊃ −4.50 cyl, ax 180 = 20/50	Clear

36 W. S.	M 22	L. E.	Dense, central	—	2/200	4.5 mm. Penetr.	2¾ yr. postop., w. −6.50 sph ⊃ −4.00 cyl, ax 130 = 20/25+	Clear
		R. E.	Central	—	1/200 without cor.; 20/70 w. cor.	6 mm. Penetr., 5¾ yr. after op., L. E.	2½ mo. postop., w. −1.75 sph ⊃ −7.50 cyl, ax 180 = 20/40	Clear
37 A. T.	M 43	L. E.	Large cystic scar	—	5/200 w. and without cor.	6 mm. Penetr.	8 mo. postop., w. +1.75 sph ⊃ −4.00 cyl, ax 60 = 20/200	Clear; patient left country; no follow-up
		R. E.	Dense	—	5/200 without cor.; w. cont. gl. = 20/70	6 mm. Penetr., 5 mo. after op. on L. E.	3 mo. postop., w. −1.00 sph ⊃ −2.00 cyl, ax 170 = 20/50	Clear
38 M. W.	M 31	R. E.	Central, involving all layers	—	10/200 without cor.	6 mm. Penetr.	9 mo. postop., 20/20 w. cor.	Clear
39 R. Z.	M 53	L. E.	Central	—	H. M.	6 mm. Penetr.	9 mo. postop., 6/200 without cor.; no improvement w. cor.	Graft 50% cloudy; complicated mature cataract present
40 S. Z.	F 41	L. E.	Central	—	H. M. at 2 ft.	6 mm. Penetr.	5 mo. postop., H. M. at 2 ft.	Opaque graft; vascularization and anterior synechia present
		L. E.	Opaque graft	Deep, large vessels below	1/200	6 mm. Penetr., 6 mo. after 1st op., L. E.	11 mo. postop., C. F. at 3 ft. (high myopia); reads newspaper held near	1/5 of graft clear
		L. E.	Mainly in posterior layers	Two fairly large ones in opaque posterior of graft	1/200	6 mm. Penetr., 1 yr. 10 mo. after 2d op., L. E.	13 mo. postop., no improvement	Graft fairly clear 1 yr. postop.; bullous keratitis and cataract present
41 L. Z.	M 35	L. E.	Slight, central	—	20/80 w. cont. gl.	6 mm. Penetr.	3 yr. 10 mo. postop., w. −5.00 sph ⊃ −1.75 cyl, ax 40 = 20/20	Clear
42 Sr. W.	F 26	R. E.	Dense, central	—	10/500 w. cor.	6 mm. Penetr.	2 yr. postop., w. −4.50 sph ⊃ −3.50 cyl, ax 120 = 20/20−2	Clear
		L. E.	Dense, central	—	15/200 without cor.	6 mm. Penetr., 6 mo. after op., R. E.	2 yr. postop., w. −3.00 sph ⊃ −6.00 cyl, ax 47 = 20/30	Clear
43 L. H.	M 47	R. E.	Nebulous	Few, small	10/200 w. cont. gl.	6.5 mm. Penetr.	1 yr. 8 mo. postop., w. −3.50 sph ⊃ −3.00 cyl, ax 110 = 20/30	Clear
		L. E.	Faint, central	—	20/50−3 w. cont. gl.	6 mm. Pentr., 1¼ yr. after op. on R. E.	5 mo. postop., w. −1.50 sph ⊃ −6.00 cyl, ax 110 = 20/50+	Clear
44 E. G.	M 30	L. E.	Diffuse, cystic	—	4/200 w. and without cor.	6 mm. Penetr.	4 mo. postop., w. cont. gl. = 20/40−	Clear
45 E. P.	F 26	R. E.	Central	—	1/200	6 mm. Penetr.	8 mo. postop., w. +3.00 sph = 20/200−	Synechiotomy and partial iridectomy done; synechiae incompletely broken; graft opaque in superficial layers
46 D. R.	M 30	L. E.	Central	—	2/200	6 mm. Penetr.	5 mo. postop., w. −5.50 sph ⊃ −0.75 cyl, ax 180 = 20/25	Clear
47 R. A.	M 30	L. E.	Central	—	H. M.	6 mm. Penetr.	1½ mo. postop., w. ph 20/200	Clear except for small adhesion at 3 o'clock
48 L. B.	F 42	R. E.	Stellate scar	—	20/200 w. cor.	6 mm. Penetr.	2 mo. postop., w. −0.50 sph ⊃ −1.00 cyl, ax 90 = 20/80	Clear

TABLE 1.—*Results of Corneal Grafts in Keratoconus**—Continued

Case No.; Patient	Sex; Age., Yr.	Eye	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
49 M. W.	F 29	R. E.	Small, central	—	4/200	6 mm. Penetr.	4 mo. postop., w. −5.00 sph = 20/200	Fairly clear graft; lens and vitreous opacities
		L. E.	Faint, central	—	C. F. without cor.; 20/70 w. cor.	6 mm. Penetr., 10 mo. after op. on R. E.	10 mo. postop., w. cont. gl. = 20/30	Clear
50 J. M.	M 21	L. E.	Some	—	C. F.	6 mm. Penetr.	14 mo. postop., w. −5.50 sph ⊃ −6.00 cyl, ax 25 = 20/30	Clear
		R. E.	Central	—	20/100 w. and without cor.	7 mm. Penetr., 11 mo. after op. on L. E.	7 mo. postop., w. −12.00 sph ⊃ −5.00 cyl, ax 20 = 20/40	Clear; faint suture marks
51 R. E.	F 31	L. E.	Scarring at apex	—	4/200 w. cor.	6 mm. Penetr.	8 mo. postop., w. −3.00 sph ⊃ +1.50 cyl, ax 150 = 20/25–1	Clear
52 E. T.	F 43	L. E.	Central opacity	—	C. F.	6 mm. Penetr.	8 mo. postop., 20/200 w. ph	Graft clear; eye amblyopic
53 Sr. M. J.	F 40	R. E.	Central	—	2/200 without cor.; 20/200 w. cor.	6 mm. Penetr.	3 mo. postop., w. +1.00 sph ⊃ +1.00 cyl, ax 90 = 20/25	Clear
54 J. R.	M 32	L. E.	Central	—	2/200 without cor.	7 mm. Penetr.	.....	Cloudy graft; bullous keratitis and herniation of Descemet's membrane developed
		L. E.	Opaque graft	Patent vessels in graft	H. M.	7 mm. Penetr., 8 mo. after 1st. op., L. E.	5 mo. postop., 2/200	Opaque graft; iris incarceration and vascularization present; epithelial bleb developed
55 A. N.	M 34	L. E.	Faint, anterior	—	10/200 w. cont. gl.	6 mm. Penetr.	4 mo. postop., no improvement	Cloudy and bulging; patient uncooperative; to be reoperated on
56 N. S.	F 26	R. E.	Central scar	—	20/200 w. and without cor.	6 mm. Penetr.	3 mo. postop., w. −2.00 sph ⊃ −4.00 cyl, ax 10 = 20/20–3	Clear
57 A. B.	M 48	L. E.	Central nebula	—	20/200 without cor.; 20/50 w. ph	6 mm. Penetr.	3 mo. postop., w. −2.00 sph ⊃ −4.50 cyl, ax 65 = 20/50	Clear
58 M. G.	M 34	L. E.	Faint, central	—	3/200 w. cor.	6 mm. Penetr.	9 mo. postop., w. −6.00 sph ⊃ −6.00 cyl, ax 180 = 20/100	Clear
59 B. F.	M 29	L. E.	Dense systic scar	—	5/200 w. and without cor.	6 mm. Penetr.	3 mo. postop., w. +3.00 sph ⊃ −7.00 cyl, ax 170 = 20/50	Clear
60 G. H.	F 28	R. E.	Central scar	—	1/200	6 mm. Penetr.	3 mo. postop., w. −7.00 sph ⊃ −2.50 cyl, ax 145 = 20/50–1	Clear
61 B. W.	M 40	L. E.	Stellate, fairly dense cystic scar at apex	—	1/200	6 mm. Penetr.	6 mo. postop., w. −4.50 sph = 20/30–1	Clear
		R. E.	Faint, nebulous at apex of cone	—	3/200	6 mm. Penetr., 2 wk. after op. on L. E.	6 mo. postop., w. −7.00 sph = 20/30+3	Clear

62 W. B.	M 24	R. E.	Few, macular	—	20/200 without cor.	6 mm. Penetr.	6 mo. postop., w. −7.00 sph ⊃ −6.00 cyl, ax 180 = 20/30	Clear
		L. E.	—	—	20/100 without cor.; 20/70 w. cor.	6 mm. Penetr., 3 mo. after op. on R. E.	1½ mo. postop., w. −4.50 sph ⊃ −5.00 cyl, ax 20 = 20/30	Clear
63 R. R.	M 15	R. E.	6 mm. in center	—	H. M. without cor.; 10/200 (?) w. cor.	7 mm. Penetr.	3 mo. postop., w. −12.50 sph = 20/200 (approximate). The patient, who has mongolism, sees pictures clearly but is unable to read because of his low intelligence	Clear
		L. E.	Faint, central	—	2/200 without cor.; 10/200 (?) w. cor.	6 mm. Penetr., 5 mo. after op. on R. E.	1 mo. postop., w. −12.50 sph = 20/200 (approximate)	Clear
64 C. W.	M 39	L. E.	6 mm. in center	—	20/100 w. cor.	6 mm. Penetr.	4 mo. postop., w. cont. gl. = 20/20	Clear
65 C. R.	M 13	L. E.	3.5 mm., central	—	10/200 without cor.; 20/300 w. cor.	6 mm. Penetr.	3 mo. postop., w. −2.50 sph ⊃ +1.00 cyl, ax 45 = 20/40–	Clear
66 E. N.	F 42	L. E.	Central	—	3/200 w. cor.	6 mm. Penetr.	No follow-up	Clear, 2 mo. postop.
		R. E.	Central, macular	—	1/200	6 mm. Penetr.	No follow-up	Clear on discharge
67 D. W.	F 23	R. E.	Diffuse, central	—	20/50–2 w. cor.	6 mm. Penetr.	No follow-up	Clear
68 E. L.	F ..	R. E.	Central, faint	—	1/200 without cor.; 20/400 w. cor.	6 mm. Penetr.	No follow-up	Clear
69 C. B.	M 28	R. E.	Slight	—	20/300 without cor.; 20/70 w. cor.	6 mm. Penetr.	No follow-up	Clear
70 B. F.	M 33	L. E.	Diffuse, central	—	5/200 w. cor.	4.5 mm. Penetr.	4 yr. postop.	Graft remained fairly clear, but eye was enucleated because of glaucoma, 4 yr. after graft; glaucoma believed present before grafting, as keratoconus developed rapidly after severe blow on eye
71† A. A.	M 28	R. E.	Moderate	—	20/200 without cor.; 20/40 w. cor.	6.5 mm. Penetr.	2½ yr. postop., w. −2.00 sph ⊃ −5.50 cyl, ax 175 = 20/25	Clear
		L. E.	Slight apical scarring	—	Balance lens	7 mm. Penetr., 2 yr. after op. on R. E.	3 mo. postop., w. −0.50 sph ⊃ −4.50 cyl, ax 10 = 20/25	Clear
72†	M	L. E.	Central, small, nebulous	—	2/200	7 mm. Penetr.	1 mo. postop., 20/100 w. ph	Slightly cloudy
73 D. S.	M 28	R. E.	Diffuse; dense scar	Few, fine	3/200	6 mm. Penetr.	No follow-up	Clear on discharge
74 D. K.	M 24	L. E.	Mottled; apical scarring	—	20/300 without cor.	6 mm. Penetr.	No follow-up	Clear on discharge

* C. F., counts fingers; cont. gl., contact glass; gl., glasses; H. M., hand motion; penetr., penetrating; ph, pinhole; w. with.
† Not operated on by author.

TABLE 2.—*Results of Corneal Grafts in Fuchs's Dystrophy**

Case No.; Patient	Sex; Age., Yr.	Eye	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1 M. B.	M 57	L. E.	Dense, central half	—	C. F. at 6 in.	5 mm. Penetr.	6 mo. postop., C. F. at 3 ft.	Cloudy graft; anterior synechiae and vascularization
		L. E.	Central haziness	At 6 o'clock	C. F. at 2 ft.	6 mm. Penetr., 2 yr. 2 mo. after 1st op. on L. E.	1¼ yr. postop., w. +1.00 sph ⊃ −6.00 cyl, ax 5 = 20/60−1	Clear graft; immature cataract
		R. E.	Central haziness, due to epithelial edema	Few, faint peripheral	5/200 w. and without cor.	6 mm. Penetr., 10 mo. after 2d op. on L. E.	1 yr. 1 mo. postop., w. −5.50 sph ⊃ −3.00 cyl, ax 110 = 20/50+1	Clear graft; incipient cataract
2 A. M.	F 56	R. E.	Central, dense	—	C. F. at 2 ft.	6 mm. Penetr.	1¼ yr. postop., w. −4.25 sph ⊃ −2.75 cyl, ax 140 = 20/25	Clear
		L. E.	Nebulous	—	L. P.	6 mm. Penetr., 1 yr. 10 mo. after op. on R. E.	3 mo. postop., H. M.	Cloudy graft; synechiotomy 7 wk. postop. unsuccessful; 2d graft to be done
3† A. D.	M 45	L. E.	Diffuse, central, involving all layers	—	20/100 w. and without cor.	6 mm. Penetr.	1½ mo. postop., 3/200	Clear graft; eye amblyopic
4† C. C.	M 73	R. E.	Dense	—	3/200 without cor.; 5/200 w. cor.	6 mm. Lamellar	2 yr. postop., 2/200	Graft ¾ clear; anterior synechia and immature cataract present
		L. E.	Diffuse, deep	—	1/200 without cor.; 5/200 w. cor.	6 mm. Penetr., 7 mo. after op. on R. E.	1½ yr. postop., 2/200	Graft very slightly hazy, particularly in posterior layer; considerable vitreous haze
5† C. L., (sister of J. L. [6])	F 62	L. E.	Total, dense, especially in posterior layers	—	H. M.	6 mm. Penetr.	11 mo. postop., C. F. at 2 ft.	Cloudy; haziness and bullae on graft surface
6† J. L., (brother of C. L. [5])	M 54	R. E.	Scattered "snowflakes"	—	10/200 w. cor.	5.5 mm. Penetr.	2 yr. 9 mo. postop., L. P.	Graft completely cloudy; vascularization and bullae present
7 M. M.	F 47	R. E.	Dense, diffuse	—	20/200 w. cor.	4.5 mm. Penetr.	1½ yr. postop., H. M.	Cloudy
8 K. D.	F 58	R. E.	Diffuse, all layers	—	H. M. at 3 ft.	6 mm. Penetr.	3 mo. postop., w. −1.00 sph = 20/40	Clear
9 A. C.	M 57	R. E.	Diffuse	—	20/400	8 mm. Lamellar	4 mo. postop., no improvement	Graft sloughed out 10 days postop., during cortisone therapy
		R. E.	Central leucoma	Few at 7 o'clock; 2 at 10 o'clock	H. M.	7 mm. Pentr., 1 yr. 8 mo. after 1st op.	3 mo. postop., no improvement	Bulging of graft and vascularization present 3 mo. postop., eventual reoperation considered
10‡ M. C.	F 56	R. E.	Central	—	H. M. without cor.; 20/400 w. cor.	6 mm. Penetr.	9 mo. postop., w. −10.50 sph ⊃ −2.50 cyl, ax 135 = 20/50	Cloudy; dystrophy entering graft; vision blurring; eye more sensitive to light
11 K. H.	F 53	R. E.	Diffuse	—	H. M.	6 mm. Penetr.	2 yr. 4 mo. postop., no improvement	Graft opaque; bullous keratitis in central portion of graft

* C. F., counts fingers; H. M., hand motion; L. P., light perception; penetr., penetrating; w. with.
† Atypical Fuch's dystrophy.
‡ Not operated on by the author.

chemical burn as one of only two diagnoses in which the outcome of a corneal graft can be predicted. Owens and associates, however, report clear grafts in 20.8% of 24 cases.[9]

The current series includes eight cases of chemical burn given adequate follow-up. In none was there a clear result (Table 9). In two cases of sulfur dioxide burn, lamellar grafts afforded relief of symptoms, although the transplants clouded. Of two lime burn cases, the graft was partly clear 16 months after operation in one and enucleation was necessary in the other.

The results were much better in the 11 cases of explosion injury. One graft was clear on discharge, with no follow-up. Ten grafts were followed for two months or longer, 7 of these remaining clear. In three of the successful grafts, the original injury was caused by a dynamite explosion; in two, by powder burn; in one, by a laboratory explosion (the eye was aphakic), and in one, by a hot lead explosion.

Of five cases of traumatic injury, clear grafts were obtained in two and cloudy results in two, while there was one enucleation, four years postoperatively.

In the group as a whole, 23 grafts had two months or more of follow-up, 9 of these, or 39.1%, remaining clear and 12 clouding, while enucleations were done in 2 instances.

Adherent Leucoma.—Whether or not adherent leucoma lends itself to keratoplasty is a matter of controversy. My series includes 17 grafts done for adherent leucoma and anterior staphyloma, and followed two months or longer. Six, or 35.3%, of these remained clear. The four cases of anterior staphyloma in this group all had a cloudy result, although in one case the graft made it possible to save the eye.

Castroviejo [3] puts adherent leucoma in his less favorable category; he considers keratoplasty contraindicated in corneal opacities with extensive anterior synechias, an opinion also expressed by Elschnig.[6] None of the cases in Stansbury's series with preoperative anterior synechias showed visual improvement following a graft. He terms eyes with wide anterior synechias unfavorable for keratoplasty and considers the operation contraindicated in adherent leucoma, citing the obstinacy with which this condition persists.[16] Owens, who terms the presence of anterior synechias one of the most difficult problems in corneal grafting, reports 6 clear grafts among 39 cases with preoperative anterior synechias.[9]

Fuchs's Dystrophy.—Until recently the patient with Fuchs's dystrophy seemed a most unlikely candidate for a corneal graft. Castroviejo [3] listed the condition as not improvable by keratoplasty. Stansbury puts this dystrophy, together with calcareous degeneration, primary fatty degeneration, and band keratitis, in the unfavorable column.[16] He adds that transplantation is probably contraindicated in these corneal degenerations, since the graft, even though technically successful, will be surrounded postoperatively by degenerative corneal tissue, on which it must depend for nutrition. The 10 grafts done for Fuchs's dystrophy in the series of Owens and associates had poor results, and the authors concluded that keratoplasty was contraindicated in this condition.[9] But, in 1952, Stocker [18] demonstrated two clear grafts in cases of this dystrophy. Keratoplasty should be done only in selected cases, he writes, in which there is considerable disease in the central portion of the cornea and a fair amount of healthy endothelium in the peripheral parts.

TABLE 3.—*Results of Corneal Grafts in Groenouw's Dystrophy**

Case No.; Patient	Sex; Age, Yr.	Eye	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1 F. K.	F 55	R. E.	Deep opacities	—	H. M.	6 mm. Penetr.	1 yr. 5 mo. postop., w. +1.75 sph ⊃ +1.50 cyl, ax 90 = 20/70	Clear
		L. E.	Leucomatous scarring	—	H. M.	7 mm. Penetr., 14 mo. after op. on R. E.	2 mo. postop., no improvement	Graft clear; cataract present
2 A. P.	F 51	L. E.	Dense, central, practically full thickness	—	10/200 without cor.	6 mm. Penetr.	2 y. 10 mo. post., w. −14.50 sph ⊃ −2.00 cyl, ax 45 = 20/40	Clear
		R. E.	Through entire thickness	—	20/200 w. and without 1 yr. after op. on L. E.	5 mm. Penetr.	7 mo. postop.	Clear
3 R. R. (sister of L. B. [4])	F 42	L. E.	Moderate, scattered	—	1/200	6 mm. Penetr.	2 yr. postop., w. +3.75 sph ⊃ −5.75 cyl, ax 25 = 20/80+3	Clear
		R. E.	Dense	—	H. M.	6 mm. Penetr., 2 yr. 2 mo. after op. on L. E.	6 mo. postop., no improvement	Graft opaque and vascularized
4 L. B. (sister of R. E. [3])	F 47	R. E.	Dense, total	Few	2/200	6 mm. Penetr.	3 mo. postop., 20/70 w. cor.	Clear
		L. E.	General cloudiness	Few	H. M.	6 mm. Penetr., 3 mo. after op. on R. E.	2 mo. postop., 20/50—1 w. cor.	Clear
5 A. G. (sister of H. J. [6] and P. McB. [7])†	F 38	L. E.	Nodular	—	20/400 w. and without cor.	6 mm. Penetr.	.....	Graft opaque and blood-stained after synechiotomy, 1¾ yr. after graft
		R. E.	Nodular	—	20/200 w. and without cor.	6 mm. Penetr., 8 mo. after op. on L. E.	1¾ yr. postop., w. −6.00 sph ⊃ −7.00 cyl, ax 180 = 20/30	Clear
		L. E.	Opaque, blood-stained graft	—	H. M.	6 mm. Penetr., 2 yr. after 1st op. on L. E.	1½ mo. postop., C. F. at 5 ft.; no improvement w. gl.	Fairly clear
6 H. J. (sister of A. G. [5] and P. McB. [7])	F 46	R. E.	Diffuse, nodular, involving all layers	—	2/200	6 mm. Penetr.	1½ mo. postop., w. −4.75 sph ⊃ −4.50 cyl, ax 45 = 20/50	Clear
		L. E.	Large, central	—	H. M.	6 mm. Penetr., 4 mo. after op. on R. E.	4 mo. postop., no improvement	Bullous keratitis and vascularization present; to be reoperated on
7 P. McB. (sister of A. G. [5] and H. J. [6])	F 40	R. E.	Diffuse, nodular, involving all layers	—	3/200 w. and without cor.	7 mm. Penetr.	3½ mo. postop., w. −3.00 sph ⊃ −3.00 cyl, ax 180 = 20/30	Clear
8 E. C.	F 41	R. E.	"Snowflake," mostly anterior ⅓; some in posterior ⅓	—	20/100 w. and without cor.	7 mm. Penetr.	2 mo. postop., w. −3.00 sph ⊃ +1.00 cyl, ax 165 = 20/70	Graft clear; anterior synechia present above

9 W. G.	M 32	L. E.	Dense, central	—	8/200 w. cor.	6 mm. Penetr.	3 yr. 5 mo. postop., w. +0.50 sph ⊃ —4.00 cyl, ax 150 = 20/30	Clear
		R. E.	Diffuse	—	20/100 w. and without cor.	6 mm. Penetr., 2 yr. after op. on L. E.	1 yr. 5 mo. postop., 20/200, no improvement	Graft cloudy because of iris adhesion along upper margin
10 C. B.	F 70	L. E.	Nebulous, central macula	Old	20/200 w. and without cor.	5 mm. Penetr.	3 mo. postop., w. —1.75 sph ⊃ —1.00 cyl, ax 90 = 20/100	Graft clear; immature cataract present
11 J. R.	M 59	L. E.	Very dense, central	—	6/200	5.5 mm. Penetr.	4 yr. postop., w. —8.00 sph ⊃ —6.00 cyl, ax 175 = 20/100—	Clear
		R. E.	Moderate, diffuse	—	6/200 without cor.	6 mm. Pentr., 1 yr. after op. on L. E.	3 yr. postop., w. —4.00 sph ⊃ —0.75 cyl, ax 10 = 20/40—	Clear
12 J. S.	M 40	R. E.	Diffusely cloudy	—	20/400 without cor.; 20/300 w. ph.	6 mm. Penetr.	1 yr. 5 mo. postop., w. —9.50 sph ⊃ —2.50 cyl, ax 128 = 20/20—2	Clear
		L. E.	Deep, central	—	6/200 without cor.	6 mm. Penetr., 1 yr. 2 mo. after op. on R. E.	3 mo. postop., w. —1.50 sph = 20/45	Clear
13 J. Sp.	F 22	R. E.	Multiple epithelial and subepithelial infiltrates, centrally	—	20/100 w. and without cor.	6 mm. Penetr.	3 mo. postop., C. F. at 2 ft.	Cloudy
		R. E.	Clouded graft	Some	C. F. at 2 ft.	6 mm. Penetr., 3½ mo. after 1st op. on R. E.	8 mo. postop., 10/200; no improvement w. gl. 10 mo. postop., w. —7.00 sph ⊃ —6.00 cyl, ax 10 = 20/70	Cloudy
14 A. L.	M 49	R. E.	Ectasia of cornea	—	2/200	7 mm. Penetr.	1 yr. 5 mo. postop., w. cont. gl. = 20/100	Graft clear; incipient cataract present
		L. E.	Entire cornea	—	20/100 w. cor.; 10/200 without cor.	6 mm. Penetr., 2⅔ yr. after op., R. E.	8 mo. postop., w. —1.50 sph ⊃ —2.75 cyl, ax 40 = 20/20—3	Clear
15 I. R.	M 60	R. E.	All layers; apparent endothelial edema	—	5/200 w. and without cor.	6 mm. Penetr.	3 mo. post op., w. —2.50 sph ⊃ —2.50 cyl, ax 180 = 20/70	Clear
		L. E.	General haze, all layers; endothelial edema	—	10/200 w. cor.	6 mm. Penetr., 5 mo. after op. on R. E.	3 wk. postop., 20/70 w. ph	Clear
16‡ F. B.	F 51	L. E.	Central and peripheral	—	2/200	6 mm. Lamellar	2½ mo. postop., 20/200; no improvement	Clear
17§ N. P.	F 48	L. E.	1.5 mm., fairly dense central	Faint	10/200 without cor.; 20/200 w. cor.	5 mm. Penetr.	2½ yr. postop., w. —1.00 sph ⊃ +7.50 cyl, ax 180 = 20/50	Clear
		R. E.	Diffuse, superficial and deep	Few, fine	5/200 without cor.	6 mm. Penetr.	4 mo. postop., w. —5.00 sph = 20/100	Clear
18 E. W.	F 69	L. E.	Central, anterior	Old, atrophied	2/200 w. and without cor.	6 mm. Lamellar	14 mo. postop., w. —2.00 sph ⊃ —4.00 cyl, ax 10 = 20/200+1	Clear; eye amblyopic
19 L. M.	F 42	L. E.	Moderate, ⅝ of cornea	—	3/200	5.5 mm. Lamellar	5 mo. postop., 3/200	Clear; cataract present

* C. F., counting fingers; cont. gl., contact glass; H. M., hand motion; gl., glasses; penetr., penetrating; ph, pinhole; w., with.
† Not operated on by author.
‡ Lattice-type dystrophy.
§ Atypical dystrophy.

TABLE 4.—*Results of Corneal Grafts in Leucoma, Adherent Leucoma, and Anterior Staphyloma**

Case No.; Patient	Sex; Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1 B. D.	F 19	R. E.	Leucoma	Anterior half of stroma	Some	2/200	5 mm. Penetr.	2 yr. postop., no improvement	Clear graft; marked cosmetic improvement; eye amblyopic
2 A. J.	M 40	L. E.	Leucoma	Moderate, lower half	Lower	H. M.	5 mm. Lamellar	1½ yr. postop., 3/200; no improvement w. gl.	Graft cloudy
3 C. M.	M 45	R. E.	Leucoma	Dense	—	5/200 without gl.	6 mm. Penetr.	1 yr. postop., no improvement	Opaque; anterior synechia above
4 T. R.	M 53	R. E.	Leucoma following infection	Complete	—	L. P.	5.5 mm. Penetr.	4 yr. postop., no improvement	Opaque
		R. E.	Opaque graft	Total	—	H. M.	10 mm. Penetr., 4½ yr. after 1st op. on R. E.	8 mo. postop., w. +5.00 sph = 4/200	Partly cloudy
5 S. S.	M 36	R. E.	Leucoma, early degeneration	Dense; central faint calcareous deposits	—	15/200 w. cor.	5 mm. Penetr.	1 yr. 7 mo. postop., 20/40 w. cont. gl.	Clear
6 R. J.	M 50	R. E.	Leucoma, nystagmus	Dense above, moderate below	Fine	2/200	5.5 mm. Penetr.	.....	Uveitis and phthisis developed; eye enucleated 5 mo. postop.
7 E. B.	M 54	L. E.	Leucoma, nystagmus	Dense, ⅔ of cornea	—	1/200	5 mm. Penetr.	2 yr. postop., 4/200; no improvement w. gl.	Clear graft; poor vision results from myopia
8 H. O.	M 28	R. E.	Leucoma, traumatic	Nebulous, all layers	Hair like, entering scar	20/400 without cor.	6 mm. Penetr.	5 mo. postop., no improvement	Clear; great cosmetic improvement; eye amblyopic
9 P. R.	M 32	L. E.	Leucoma, traumatic	Dense, all layers	Some	2/200	6 mm. Penetr.	2 mo. postop., w. −1.50 sph ⌢ +4.50 cyl, ax 90 = 20/20−3	Clear
10 W. S.	M 15	L. E.	Leucoma	In pupillary area	—	2/200	6 mm. Penetr.	No follow-up	Clear on discharge
11 B. C.	F 44	R. E.	Adherent leucoma	Dense, central	Many, empty; none patent	10/200 without cor.	6 mm. Penetr.	14 mo. postop., 1/200; no improvement w. gl.	Graft partially opaque; anterior synechia present
12 D. B.	F 23	R. E.	Adherent leucoma	Faint	—	2/200 without cor.	5 mm. Penetr.	1¼ yr. postop., C. F.	Slightly hazy inferiorly; ant. synechia present
13 C. M.	M 52	R. E.	Adherent leucoma	Dense cystic scar, ant. synechia present	Several	2/200	6 mm. Penetr.; synechiotomy	2 yr. 10 mo. postop., w. −6.00 sph ⌢ −2.00 cyl, ax 30 = 20/40+2	Clear; marked cosmetic improvement
14 E. S.	F 32	R. E.	Adherent leucoma	Dense	Few, small	2/200	6 mm. Penetr.	16 mo. postop., 5/200; no improvement w. gl.	Clear; nystagmus present
15 S. K.	M 30	R. E.	Adherent leucoma	Dense, involving whole cornea	In scar	1/200	5 mm. Lamellar	4 mo. postop., 20/200; no improvement w. gl.	Graft clear; cosmetic improvement

16 N. L.	F 20	L. E.	Adherent leucoma, following	Dense; synechia present	Many, small, in scar	3/200 without cor.	6 mm. Penetr.	9 mo. postop., no improvement	Partly cloudy; marked cosmetic improvement; immature cataract present
17 N. W.	M 37	R. E.	Adherent leucoma	Dense	Peripheral	H. M.	5.5 mm. Penetr.	No follow-up	Clear 5 mo. postop.
18 O. M.	M 24	L. E.	Adherent leucoma, fine nystagmus	Complete; in lower half, anterior synechia present	Moderate	3/200 without cor.	5 mm. Lamellar	15 mo. postop., no improvement	Partly clear; great cosmetic improvement
19 A. S.	M 46	R. E.	Adherent leucoma following foreign body infection	Moderate, central; anterior synechia	One, large	H. M.	6 mm. Penetr.; tiny iridectomy	4 mo. postop., 20/200; no improvement w. gl.	Partly cloudy; marked cosmetic improvement
20 R. O.	M 22	L. E.	Adherent leucoma	Dense	Many, patent, in scar	L. P.	10 mm. Lamellar	10 mo. postop., no improvement	Graft opaque; painted cont. gl. recommended
21 J. C.	M 18	L. E.	Adherent leucoma (injury)	Central; anterior synechia	Few, fine, hair-like	2/200 w. and without cor.	6 mm. Penetr.	2 mo. postop., w. −6.00 sph ⌢ +6.00 cyl, ax 105 = 20/200	Clear except for two nebulous lines; anterior synechia
22† L. P.	M 42	L. E.	Adherent leucoma	Dense, central; anterior synechia	Small	H. M.	6 mm. Penetr.	15 mo. postop., no improvement	Translucent; many new blood vessels present; cataract
23 E. W.	F 42	L. E.	Adherent leucoma	Dense; anterior synechia	Few, fine, hair-like	4/200 w. and without cor.	6 mm. Penetr.	7 mo. postop., w. −15.00 sph ⌢ +4.50 cyl, ax 60 = 20/60−2	Clear
24 I. C.	M 37	L. E.	Scarring, cause unknown	Dense, central	—	H. M.	6 mm. Penetr.	9 mo. postop., faulty L. P.	Complete staphyloma
		L. E.	Dense; anterior staphyloma; graft protruding	Complete	In staphyloma	L. P.	7 mm. Penetr., 9 mo. after 1st op. on L. E.	4 mo. postop., L. P.	Opaque
25† C. O.	M 56	L. E.	Anterior staphyloma	Leucoma, resulting from failure of graft op. done elsewhere	Number	H. M.	10 mm. Penetr.	3 mo. postop., no improvement	Cloudy; cosmetic improvement; eye saved
26 F. A.	M 57	R. E.	Anterior staphyloma (recurrent iritis and keratitis)	Very dense, complete	Some	H. M.	5 mm. Penetr.	1 mo. postop., 4/200; no improvement w. gl.	Cloudy
		R. E.	Cloudy graft	Dense, central	Some	L. P.	5 mm. Penetr., 8 mo. after 1st op. on R. E.	1 yr. 8 mo. postop., no improvement	Cloudy; anterior synechia and vascularization
27 M. L.	F 54	L. E.	Anterior staphyloma	Very dense, central	Some, fine	H. M.	5 mm. Penetr.	1¾ yr. postop., L. P.	Cloudy graft; glaucoma developed

* Cont. gl., contact glasses; gl., glasses; H. M., hand motion; L. P., light perception; pentr., penetrating, and w. with.
† Not operated on by author.

TABLE 5.—*Results of Corneal Grafts in Keratitis*[a]

Case No.; Patient	Sex; Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1 C. D.	M 29	R. E.	Old interst. kerat.	Diffusely hazy, all layers	Several ghost vessels	H. M.	6 mm. Penetr.	No follow-up	Clear on discharge
2 E. B.	F 61	R. E.	Old interst. kerat.	Very dense, central	Tiny, peripheral	2/400	6 mm. Penetr.	3 yr. 8 mo. postop., w. −6.00 sph = 5/200	Clear; poor vision due to myopic degeneration
		L. E.	Old interst. kerat.	Nebulous	Faint ghost	5/200 w. cor.	6 mm. Lamellar and iridectomy, 4 yr. after op. in R. E.	1 yr. postop., w. +3.50 cyl, ax 160 = 10/70	Clear; glaucoma present preop.
3 C. S.	F 39	R. E.	Old interst. kerat.	Dense, below	Ghost	3/200	6 mm. Penetr.	2¼ yr. postop., w. +1.50 sph = 20/200	Clear; poor vision due to myopic choroiditis
		L. E.	Old interst. kerat.	Diffuse, all layers	Ghost	H. M.	6.5 mm. Penetr.	2 mo. postop., no improvement	Clear; cataract present
4 M. R.	F 50	L. E.	Old interst. kerat.	Dense, central	Few, faint, peripheral	H. M.	5 mm. Penetr.	22 mo. postop., no improvement	Lower ½ of graft opaque; deep vascularization
5 E. M.	F 20	L. E.	Old interst. kerat.	Moderate	Slight	10/200 without cor.	4.5 mm. Penetr.	7 mo. postop., 20/200; no improvement w. gl.	Cloudy; vascularization
		L. E.	Cloudy graft	Dense, central	Slight	20/200 without cor.	4.5 mm. Penetr., 7 mo. after 1st op. on L. E.	2½ yr. postop., 4/200; no improvement w. gl.	Graft cloudy; vascularization
		L. E.	Cloudy graft	Dense, macula	Many fine	4/200 without cor.	5 mm. Penetr., 2½ yr. after 2d op. on L. E.	5 yr. postop., 20/40 w. cont. gl.	Clear
		R. E.	Old interst. kerat.	Dense	Ghost	10/200 without cor.	6 mm. Lamellar, 3 yr. after 3d op. on L. E.	16 mo. postop., 5/200	Graft opaque in nasal portion; vascularization
		R. E.	Cloudy graft	Central, dense	Several, below	5/200	6 mm. Penetr., 16 mo. after 1st op. on R. E.	No follow-up	Clearing, 9 mo. postop.
6 M. M.	F 46	L. E.	Old interst. kerat. (recurrent ulcers)	Nebulous	Few, superficial, hair-like	10/200 without cor.	5 mm. Lamellar	3½ yr. postop., w. −6.00 syph ⊃ −4.50 cyl, ax 5 = 20/100	Clear
		R. E.	Same as in L. E.	Nebulous	Few, fine, hair-like	20/100 w. cor.	6 mm. Penetr., 2 yr. 8 mo. after op. on L. E.	10 mo. postop., w. −6.00 sph ⊃ −8.00 cyl, ax 95 = 20/100	Clear; eye amblyopic
7 E. O.	F 28	L. E.	Old interst. kerat.	Macula, lower half	Deep, below and temporally	20/200 without cor.	6 mm. Penetr.	1 yr. postop., no improvement	Cloudy
		R. E.	Same as in L. E.	Macula	Few, old	20/70 w. cor.	5 mm. Penetr.	2 yr. 2 mo. postop., 20/40 w. cor.	Clear
8 F. M.	F 49	L. E.	Interst. kerat.	Densely cloudy	Number, deep, hair-like	3/200 w. cor.	6.5 mm. Lamellar	14 mo. postop., w. +1.50 sph ⊃ +1.50 cyl, ax 90 = 20/100	Clear
		R. E.	Interst. kerat.	Diffuse, nebulous, all layers	Many, old, hair-like	5/200 without cor.; 20/200 w. cor.	6 mm. Penetr., 10 mo. after op. on eye	4 mo. postop., w. −3.50 sph ⊃ −3.00 cyl, ax 45 = 20/70	Clear
9 F. C.	F 39	R. E.	Interst. kerat.	Diffuse scarring	Many	C. F.	5 mm. Penetr.	5 mo. postop., no improvement	Cloudy

		R. E.	Cloudy graft	Diffuse	Ghost	L. P.	5 mm. Penetr., 7 mo. after 1st op. on R. E.	6 yr. postop., no improvement	Cloudy; vascularization; to be reoperated on
		L. E.	Interst. kerat.	Diffuse	Many	2/200	6 mm. Penetr., 3 yr. after 2d op. on R. E.	.....	Vascularization developed, with iris incarceration; eye enucleated, 1 yr. 5 mo. postop.
10	F 56	L. E.	Interst. kerat.	Central, superficial and deep	Ghost	2/200 without cor.; 10/200 w. cor.	5 mm. Penetr.	1 yr. postop., no improvement	Clear; incipient cataract present
11† M. M.	F 56	R. E.	Old interst. kerat.	Central, macula	Few, marginal	20/200 w. cor.	6 mm. Penetr.	No follow-up	Hazy 4 mo. postop.; no vascularization
12 C. F.	F 55	L. E.	Interst. kerat.	Dense	Number	H. M.	4.5 mm. Penetr.	6 mo. postop., no improvement	Opaque; some vascularization at periphery (keratoconus present preop.)
13 J. K.	F 22	L. E.	Interst. kerat.	Nebulous	Many, superficial	4/200 without cor.	6 mm. Penetr.	1 yr. postop., no improvement	Clouded; anterior synechia present; to be reoperated on
14	F	L. E.	Interst. kerat.	Nebulous all	Many, atrophied	2/200 w. and without cor.	6 mm. Penetr.	5 mo. postop., w. −8.00 sph = 20/100+1	Clear; myopic choroiditis present
		R. E.	Interst. kerat.	Nebulous, diffuse	Many, superficial, ghost	3/200 without cor.	6 mm. Penetr., 1 yr. 5 mo. after op. on L. E.	No follow-up	Clear, 2 mo. postop.
15† F. L.	M 25	R. E.	Interst. kerat.	Central	Ghost	20/500 w. cor.	7 mm. Penetr.	No follow-up	Clear on discharge
16 R. M.	M 67	R. E.	Interst. kerat.	Central, moderately dense	Deep, fine	20/70 w. cor.	6 mm. Penetr.	4 mo. postop., w. −0.50 sph ⊃ +3.50 cyl, ax 75 = 20/30	Clear
17 F. G.	F 44	L. E.	Interst. kerat.	Diffuse	Ghost	3/200	5 mm. Penetr.	5 yr. postop., L. P.	Opaque; vascularization
18 J. C.	F 30	R. E.	Interst. kerat.	Nebulous	Few, hair-like	2/200 without cor.	6 mm. Penetr.	2 yr. postop., w. +0.75 sph ⊃ −2.50 cyl, ax 23 = 20/25	Clear
19 E. B.	F 61	R. E.	Interst. kerat.	Very dense, central	Tiny, peripheral	2/400 w. cor.	6 mm. Penetr.	3½ yr. postop., w. −6.00 sph = 5/200	Clear; poor vision due to myopic degeneration (glaucoma present preop.)
		L. E.	Interst. kerat.	Nebulous	Faint, ghost	5/200 w. cor.	6 mm. Lamellar and iridectomy 4 yr. after op. on R. E.	1 yr. postop., w. +3.50 cyl, ax 160 = 10/70	Clear
20 M. W.	M 43	L. E.	Interst. kerat. and uveitis	Very dense, diffuse	Slight	20/200 w. cor.	4.5 mm. Penetr.	6 yr. 9 mo. postop., w. −1.50 sph ⊃ −0.75 cyl, ax 70 = 20/20—3	Clear
		R. E.	Same as in L. E.	Dense, central ⅔	Many, small	20/200 w. cor.	5 mm. Penetr., 1 yr. 8 mo. after op. on L. E.	5 yr. postop., w. −0.75 sph ⊃ +1.75 cyl, ax 180 = 20/40	Clear
21 M. T.	F 55	R. E.	Old interst. kerat.	Nebulous	Ghost	2/200 w. and without cor.	6 mm. Penetr.	1 yr. postop., w. cont. gl. = 10/200	Clear except for haze in Descemet's membrane, 1 to 4 o'clock; poor vision due to atrophy of macular area
		L. E.	Old interst. kerat.	Central, nebulous	Ghost	20/200 w. cor.	6 mm. Penetr., 4 mo. after op. on R. E.	5 mo. postop., w. −3.00 sph = 20/50	Clear

TABLE 5.—*Results of Corneal Grafts in Keratitis**—Continued

Case No.; Patient	Sex; Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
22 R. G.	F 17	R. E.	Interst. kerat.	Central areas of degeneration	Old, deep; several new, superficial	20/100—1 without cor.	5 mm. Lamellar	4 mo. postop., 20/100; no improvement w. gl.	Cloudy
		R. E.	Cloudy graft	Dense	Deep	20/100 without cor.	6 mm. Penetr., 4 mo. after 1st op. on R. E.	1 mo. postop., w. +1.50 sph ⊃ +4.50 cyl, ax 65 = 20/40	Graft subsequently clouded, with recurrence of interst. kerat.
23† C. P.	F 35	R. E.	Interst. kerat.	Dystrophic epithelial changes	Throughout	3/200	5.5 mm. Penetr.	5 mo. postop., L. P.	Cloudy
24 W. R.	M 22	R. E.	Interst. kerat.	Central, macula	Ghost	1/200	5.5 mm. Penetr.	3 yr. 4 mo. postop., 3/200; no improvement w. gl.	Clear; poor vision, due to optic atrophy
25 J. A.	M 46	L. E.	Interst. kerat.	Diffuse, all layers	Ghost	3/200	6 mm. Penetr.	4 mo. postop., w. −6.00 sph ⊃ −4.00 cyl, ax 140 = 20/100	Slight haziness; patient can read small print
26† G. B.	F 52	L. E.	Interst. kerat.	Through full thickness	Few; ghost	3/200 without cor.	6 mm. Penetr.	2 mo. postop., L. P.	Opaque and vascularized
27 S. J.	F 30	L. E.	Interst. kerat.	Dense	Several, tiny	20/200 w. cor.	5 mm. Penetr.	2 mo. postop., w. −2.00 sph ⊃ −3.00 cyl, ax 135 = 20/50+	Clear
28 R. N.	M 37	L. E.	Interst. kerat.; early calcareous degeneration	Diffuse; posterior synechia	Ghost	20/200 without cor.	5 mm. Penetr.	5 yr. postop., w. −5.00 cyl, ax 180 = 20/200+	Clear
		R. E.	Interst. kerat.	Band keratitis, ⅔ of anterior stroma	Old	20/200 without cor.; 20/70 w. cor.	6 mm. Lamellar, 5½ after op. on L. E.	No follow-up	Clear; cataract and iris adhesion present 5 mo. postop.
29 M. T.	F 38	L. E.	Interst. kerat.	Translucent centrally	Ghost-like, peripheral, above and temporal	H. M.	6 mm. Penetr.	5 mo. postop., w. +12.00 sph ⊃ +1.00 cyl, ax 90 = 20/50	Clear
30 C. H.	F 17	R. E.	Interst. kerat.	Dense, central	Ghost	10/200 without cor.	6 mm. Penetr.	No follow-up	Clear, 2 mo. postop.
31 R. M.	F 35	R. E.	Interst. kerat. and uveitis	Central, all layers; leucomatous	Few ghost	2/200	6 mm. Penetr.	1½ mo. postop., w. −3.00 sph ⊃ +4.00 cyl, ax 30 = 20/50	Clear
32 L. K.	M 51	L. E.	Interst. kerat.	Dense, ¾	Many	20/70 without cor.	5 mm. Penetr.	8 mo. postop., 3/200	Cloudy
		L. E.	Cloudy graft	Generalized; most marked below	Deep, small, around graft	3/200	6 mm. Penetr.	1 yr. 7 mo. postop., w. −10.00 sph = 20/500	Semiopaque
33 Y. P.	F 66	R. E.	Interst. kerat.	Nebulous	Few	1/200	5 mm. Penetr.	4 mo. postop., H. M.	Cloudy; glaucoma developed
		L. E.	Interst. kerat.	Nebulous	Few	1/200	5 mm. Penetr., 6 mo. after op. on R. E.	15 mo. postop., w. +1.50 sph ⊃ +4.50 cyl, ax 180 = 3/200	Cloudy; glaucoma developed
34† A. H.	F 27	R. E.	Interst. kerat.	Diffuse	Few, gross	6/200 without cor.	6 mm. Penetr.	15 mo. postop., 10/200 w. cor.	Cloudy; vascularization; to be reoperated on
35 M. P.	F 37	R. E.	Old interst. kerat.	Central	Present	H. M.	5 mm. Lamellar	6 mo. postop., no improvement	Cloudy; adherent leucoma below
		R. E.	Cloudy graft	Adherent leucoma below	Up to graft	H. M.	6 mm. Lamellar	7 mo. postop., no improvement	Opaque; graft done preparatory to penetr.

		R. E.	Opaque graft (chronic keratitis)	Opacity	Growing to graft	H. M.	7 mm. Penetr.	3 mo. postop., H. M.	Cloudy
36 L. S.	F 56	L. E.	Old keratitis	Diffuse, macular, all layers	Old, empty	1/200 without cor.	6 mm. Penetr.	1 yr. postop., 3/200; no improvement w. gl.	Clear; poor vision, due to large chorioretinal scar
37 M. B.	M 72	R. E.	Keratitis	Central	Few, fine, from above	L. P.	6 mm. Lamellar	8 mo. postop., no improvement	Cloudy graft; vascularization; subjective and objective improvement
38 M. S.	M 53	R. E.	Active keratitis	Diffuse, throughout	—	L. P.	5 mm. Lamellar	5 mo. postop., no improvement	Cloudy; graft done for therapeutic purposes
39 A. B.	F 63	L. E.	Old keratitis (scarring)	Mottled, extending deep into stroma	—	H. M.	6 mm. Lamellar	No follow-up	Graft lost 1 mo. postop., subjective improvement
40† E. S.	M 56	L. E.	Old keratitis (scarring)	Dense, central	Few, atrophic; few patent	H. M.	6 mm. Penetr.	5 mo. postop., w. −2.00 sph, ax 10 = 20/50	Clear
41 M. W.	F 49	R. E.	Old band-shaped keratitis	Diffuse, all layers	Few, peripheral	H. M.	6 mm. Lamellar	9 mo. postop., no improvement	Slight, mottled haze in graft and underlying cornea
42 C. B.	F 70	L. E.	Band-shaped keratitis	Leucoma	Few, small	H. M.	6 mm. Penetr.	2 mo. postop., 10/200; no improvement w. gl.	Clear; incipient cataract
		R. E.	Band-shaped keratitis	Band opacities	—	H. M.	6 mm. Penetr., 4 mo. after op. on L. E.	1½ mo. postop., 6/200; no improvement w. gl.	Clear; cataract present
43 C. H.	F 63	L. E.	Band-shaped keratitis	Diffuse	—	L. P.	6 mm. Lamellar	2½ mo. postop., w. −6.50 sph = 20/200	Graft slightly hazy, 14 mo. postop.
		R. E.	Band-shaped keratitis	Band opacities	—	6/200 without cor.; 20/70 w. cor.	6 mm. Penetr., 4 mo. after op. on L. E.	4 mo. postop., H. M.	Cloudy

TABLE 5.—*Results of Corneal Grafts in Keratitis**—Continued

Case No.; Patient	Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperation Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
44‡ C. M.	M 64	R. E.	Bullous keratitis	Nebulous	—	.....	4.5 mm. Lamellar	6 mo. postop., 3/200; no improvement w. gl.	Cloudy; great relief of symptoms
45‡ J. G.	M	R. E.	Bullous keratitis	Diffuse, involving whole stroma	—	H. M. without cor.; 5/200 w. cor.	6 mm. Lamellar	1 mo. postop., no improvement	Cloudy; bullous keratitis arrested
46‡ A. A.	M 66	R. E.	Bullous keratitis	Moderate, diffuse	—	20/200 without cor.	5 mm. Penetr.	2 yr. 2 mo. postop., C. F.	Cloudy; symptoms greatly relieved
47‡ C. L.	M 58	R. E.	Bullous keratitis	Diffuse haziness, all layers; increase in thickness	—	6/300 without cor.	6 mm. Lamellar	9 mo. postop., H. M. (shadowy)	Cloudy; recurring anterior vesicles (glaucoma and aphakia present preop.)
48‡ L. S.	F 66	R. E.	Bullous keratitis	Complete; anterior synechia	At limbus above	L. P.	6 mm. Lamellar	1 yr. postop., 1/200; no improvement w. gl.	Recurrence of bullous keratitis after 6 mo.
49 T. C.	F	L. E.	Sclerosing keratitis, recurrent	Dense	Fine	5/200 without cor.	6 mm. Penetr.	10 mo. postop., 3/200	Cloudy; vascularization present
		L. E.	Cloudy graft	Cloudy	Below	3/200	6 mm. Penetr.	1 yr. 9 mo. postop., w. cont. gl. = 20/30	Clear
50 I. M.	M 53	L. E.	Sclerosing keratitis, recurrent	Total	Few	6/200 without cor.	4.5 mm. Penetr.	2 yr. postop., w. −8.00 sph ⌒ −2.00 cyl, ax 175 = 20/50	Clear
		R. E.	Same as in L. E.	Dense, ¾	—	L. P.	5 mm. Penetr.	1½ yr. postop., 1/200; no improvement w. gl.	Clear except for slight clouding inferiorly
51 J. R.	M 36	L. E.	Sclerosing keratitis	Very dense	Slight	4/200	4.5 mm. Penetr.	16 mo. postop., w. −10.00 sph = 5/200	Partly clear (cataract and nystagmus present preop.)

* Interst. kerat., interstitial keratitis; pentr., penetrating; L. P., light perception; H. M., hand movements; C. F., counts fingers; gl., glasses; w. cont. gl., with contact glass.
† Not operated on by the author.
‡ Graft done for therapeutic purposes.

TABLE 6.—*Results of Corneal Grafts in Trachomatous Scarring**

Case No.; Patient	Sex; Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1 R. N.	M 57	R. E.	Trachomatous scarring	Diffuse	—	2/200 without cor.; 20/200 w. cor.	6 mm. Penetr.	9 mo. postop., no improvement	Graft slightly hazy; anterior synechia and vascularization present
2 F. McD.	M 51	L. E.	Trachomatous scarring	Very dense, lower ¾; anterior synechia	Some	2/200 without cor.	5 mm. Lamellar	1 yr. postop., no improvement	Slight clouding of graft
		L. E.	Cloudy graft; scarring	Densely opaque in deep layers; anterior synechia	Deep	1/200 without cor.	6 mm. Penetr., 13 mo. after 1st op. on L. E.	2 yr. 9 mo. postop., 10/200; no improvement w. gl.	Graft translucent; marked cosmetic improvement
		R. E.	Trachomatous scarring	Lower ½ of stoma opaque; endothelium opaque	Some	20/200 without cor.; 20/100 w. cor.	6 mm. Lamellar, 15 mo. after 2d op. on L. E.	6 mo. postop., 3/200; no improvement w. gl.	Cloudy; vascularization
		R. E.	Trachomatous scarring	Dense leucoma	Many in posterior layers	3/200 w. and without cor.	6 mm. Penetr., 6 mo. after 1st op. on R. E.	1 yr. postop., 5/200; no improvement w. gl.	Cloudy
3 W. T.	M 58	R. E.	Trachomatous scarring; uveitis	Diffuse, anterior	Few, hairlike	20/200 w. cor.	6 mm. Penetr.	5 mo. postop., no improvement	Cloudy and edematous; uveitis developed postop.
4 A. E.	M 65	R. E.	Trachomatous scarring	Moderate, central	Slight, peripheral	10/200 w. cor.	6 mm. Penetr.	2 yr. postop., w. −0.75 sph ⊃ −3.00 cyl, ax 175 = 20/60	Clear; immature cataract present
		L. E.	Trachomatous scarring	Dense, leucoma	Superficial, peripheral	L. P.	5 mm. Penetr., 1 yr. 8 mo. after op. on R. E.	5 mo. postop., 10/200, w. balance lens	Clear
5 I. H.	M 56	L. E.	Trachomatous scarring	Dense	Pannus at 12 o'clock	5/200	6 mm. Penetr.	1 yr. 8 mo. postop., L. P.	Graft hazy; myopic choroiditis present
6 L. N.	M 33	L. E.	Trachoma	Very dense	Slight	L. P.	4.5 mm. Penetr.	1 yr. postop., H. M.	Clear; poor vision due to nystagmus
7 R. A.	F 54	R. E.	Trachoma (chronic keratitis)	Central, macula	Superficial, peripheral	20/100 w. cor.	5 mm. Lamellar	7 mo. postop., no improvement	Clear
8† G. G.	F 68	L. E.	Old trachoma; keratitis	Diffuse haziness throughout	Many, tiny	20/200 without cor.	6 mm. Penetr.	2 yr. 10 mo. postop., w. +3.50 sph ⊃ −6.00 cyl, ax 180 = 20/100	Diffuse clouding of graft
9 H. H.	F 61	R. E.	Keratitis (probably trachomatous)	Diffuse, central and peripheral	Deep and superficial	5/400 without cor.	6 mm. Penetr.	11 mo. postop., no improvement	Graft clear; cataract present

* Cor., correction; gl., glasses; H. M., hand motion; L. P., light perception; penetr., penetrating.
† Not operated on by the author.

TABLE 7.—*Results of Corneal Grafts in Active Ulcers and Ulcer Scarring**

Case No.; Patient	Sex; Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1† J. M.	M 48	L. E.	Mooren's ulcer	Dense, peripheral	Number	H. M.	5 mm. Lamellar	3 yr. postop., H. M.	Opaque; ulcer arrested; penetrating graft to be done eventually
2† M. R.	F 45	R. E.	Mooren's ulcer	3 to 11 o'clock	—	20/100 without cor.	6 mm. Lamellar	2 mo. postop., 5/100; no improvement w. gl.	Clear; iris adhesion
3† H. G.	F 47	R. E.	Scar from active herpetic ulcer	Dense, 2 anterior synechiae	Present	3/200 w. and without cor.	6 mm. Lamellar	8 mo. postop., no improvement	Cloudy; enucleation avoided
4† D. B.	F 53	R. E.	Herpetic ulcer	All layers	Many; hairlike	H. M.	4.5 mm. Lamellar	14 mo. postop., 2/200; no improvement w. gl.	Cloudy; operation done to stop spread of ulcer
5† J. C.	M 52	L. E.	Ulcer, recurrent	Central macula	Several	4/200 without cor.; 20/100 w. ph.	6 mm. Penetr.	4 mo. postop., w. −1.50 sph ⌒ −3.50 cyl, ax 70 = 20/100	Clear; incipient cataract
6† J. S.	F 55	L. E.	Ulcer, recurrent	Dense, central	Several	3/200	5 mm. Penetr.	5 yr. 3 mo. postop., w. +4.00 cyl, 00 = 20/70	Partly clear
7 G. F.	F 67	L. E.	Ulcer	Very dense, central	—	3/200 without cor.	5 mm. Penetr.	9 mo. postop., w. +6.00 sph = 20/60	Clear
8 J. H.	M 75	L. E.	Ulcer	Marked scarring	—	H. M.	5 mm. Square, penetr.	15 mo. postop., no improvement	Cloudy; glaucoma
9 M. W.	F 29	L. E.	Ulcer	Very dense, central	Many, small	2/200 without cor.	4.5 mm.	8 mo. postop., no improvement	Clear; cosmetic improvement
10 M. W.	F 58	L. E.	Ulcer	Dense, central; moderate in rest of cornea	Number, deep	5/200	4.5 mm. Penetr.	1 yr. postop., no improvement	Clear, 1 yr. postop., cataract present
11 J. H.	M 44	R. E.	Ulcer scar	Dense, diffuse; anterior synechia	—	6/400 w. cor.	5 mm. Penetr.	2¾ yr. postop., no improvement	Cloudy; vascularization and anterior synechia
12 M. V.	F 55	L. E.	Old ulcer scar	Very dense; diffuse synechiae and many old K. P.	Below, at 6 o'clock	L. P.	5 mm. Penetr.	4 mo. postop., no improvement	Slightly cloudy; vascularization
13 M. Cs.	F 70	R. E.	Ulcer scar	Central, diffuse, nebulous, all layers	—	2/200 w. and without cor.	6 mm. Penetr.	6 mo. postop., w. +9.50 sph ⌒ +1.50 cyl, ax 90 = 20/200	Graft clear 8 mo. postop.; vitreous cloudy
14 C. B.	M 41	L. E.	Ulcer scarring	Diffuse	—	5/400 without cor.	6 mm. Penetr.	2½ yr. postop., w. cont. gl. = 20/20	Clear
		R. E.	Ulcer scarring	Dense, diffuse	—	20/200 without cor.	5 mm. Lamellar, 2 mo. after op. on L. E.	3 mo. postop., 20/200; no improvement w. gl.	Cloudy; vascularization

		R. E.	Cloudy graft	Dense leucoma	Deep, 3-9 o'clock	20/200 w. and without cor.	5.5 mm. Penetr., 3 mo. after 1st op. on R. E.	3 mo. postop., no improvement	Partly cloudy; peripheral vascularization
		R. E.	Cloudy graft	Slight clouding temporally	Few, peripheral	Balance lens	5 mm. Penetr., 5 mo. after 2d op. on R. E.	1 yr. 8 mo. postop., w. cont. gl. = 20/20	Clear
15 P. B.	M 64	L. E.	Ulcer scar	Leucoma	Few, hair-like	1/200	5.5 mm. Penetr.	1 yr. postop., no improvement	Clear; cataract present
16 A. C.	M 54	R. E.	Old ulcer scar	Central	—	20/200 w. ph.	5 mm. Penetr.	1½ mo. postop., w. +2.75 cyl, ax 87 = 20/100; at times, 20/70	Clear, 4 mo. postop.
17 O. D.	M 61	R. E.	Old ulcer scar	Central, all layers	Few	2/200	6 mm. Penetr.	10 mo. postop., H. M.	Graft scarred after panophthalmitis; vascularization
		R. E.	Scarred graft	Very dense	Peripheral	H. M.	5 mm. Penetr., 10 mo. after 1st graft in R. E.	4 mo. postop., balance lens	Cloudy, 2 yr. postop.; to be reoperated on
18‡ J. B.	F 57	R. E.	Ulcer scar	Dense, central	Many	L. P.	7 mm. Lamellar	9 mo. postop., no improvement	Cloudy; op. preparatory to penetr. graft
19 H. C.	M 23	L. E.	Macula	Middle layer	One large; several small, in stroma	5/200	6 mm. Lamellar	No follow-up	Clear on discharge
20 J. H.	M 52	R. E.	Macula	Less than ½ cornea	—	20/200 without cor.	5 mm. Penetr.	6 mo. postop., w. +0.75 sph ⊃ −1.50 cyl, ax 90 = 20/70	Clear
21 H. F.	F 44	L. E.	Macula	Nebulous; central scar	Minute, ghost	20/200 w. and without cor.	6 mm. Lamellar	2 mo. postop., no improvement	Clear; preparatory to penetr. graft
		L. E.	Nebula beneath lamellar graft	Nebulous	One patent; several ghost	20/200 without cor.	7 mm. Penetr., 5 mo. after 1st op. in L. E.	1½ mo. postop., w. −3.50 sph ⊃ −8.75 cyl, 135 = 20/50	Clear
22 T. M.	F 45	R. E.	Macula	Scarring	—	20/200 w. cor.	6 mm. Penetr.	14 mo. postop., w. −6.00 sph ⊃ +1.75 cyl, ax 130 = 20/40	Clear
23 O. B.	M 61	R. E.	Macula	Central	—	20/100 w. cor.	6 mm. Penetr.	2 mo. postop., no improvement	Clear; cataract
24 M. C.	F 40	R. E.	Macula	Fairly dense, lower ½	Few, small, lower half	5/200 without cor.	5 mm. Penetr.	14 mo. postop., no improvement	Hazy; to be reoperated on
		L. E.	Macula	Throughout	Lower half	5/200 w. and without cor.	5 mm. Penetr.	1 yr. postop., w. −3.00 sph ⊃ −5.00 cyl, ax 135 = 20/70	Clear

* Penetr., penetration; H. M., hand movements; L. P., light perception; w. gl., with glasses; cont. gl., contact glass; ph., pinhole.

† Done for therapeutic purposes.

‡ Not operated on by author.

TABLE 8.—*Results of Corneal Grafts in Scarring (Nonspecific)**

Case No.; Patient	Sex; Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1 K. W.	M 56	L. E.	Scar	Surface opaque and thickened	—	H. M.	7 mm. Lamellar	No follow-up	Graft translucent, 3 wk. postop.
2 M. S.	F 46	L. E.	Scar	All layers	Central	4/200	5 mm. Penetr.	1 yr. postop., 6/200; no improvement w. gl.	Clouded, 2 yr. postop.; vascularization and bullous keratitis developed
3 C. D.	F 40	R. E.	Scar	Dense, lower central	Several, superficial	1/200	5 mm. Penetr.	5 wk. postop., w. −3.00 sph ⊃ +3.00 cyl, ax 90 = 10/50	Partly cloudy; a few vessels from above
4 K. C.	F 46	R. E.	Scar	Mostly central	Old	L. P.	6 mm. Penetr.	2 mo. postop., no improvement	Clear, amblyopia ex anopsia
5 C. B.	M 48	R. E.	Scarring	Dense, lower ⅔	Many deep	4/200 without cor.	6 mm. Lamellar	10 mo. postop., 20/100; no improvement w. gl.	Partly cloudy
6 G. P.	F 34	R. E.	Scarring	Dense, all layers	—	6/200 w. cor.	6 mm. Penetr.	1 yr. 9 mo. postop., w. −2.50 sph ⊃ −5.00 cyl, ax 15 = 20/70	Clear
7 A. L.	F 54	R. E.	Scar	Dense, central	Several, large, temporally	20/400 w. cor.	6 mm. Penetr.	2 mo. postop., w. −3.50 cyl, ax 90 = 20/50	Clear
8 D. L.	M 21	L. E.	Scarring	Moderate, superior	—	10/200 w. cor.	6 mm. Penetr.	5 mo. postop., w. −4.00 cyl, ax 175 = 20/30	Clear
9 L. M.	M 39	R. E.	Scars	Moderate, central	—	20/400	5 mm. Penetr.	4 yr. 4 mo. postop., w. −16.00 sph ⊃ −4.00 cyl, ax 165 = 20/300	Clear; eye amblyopic
10 M. M.	F	L. E.	Scars	Pericentral	—	20/400	5 mm. Penetr.	7 mo. postop., w. −4.5 sph ⊃ −3.25 cyl, ax 85 = 20/60	Clear; incipient cataract
11 C. N.	F 67	R. E.	Scars	Central, dense, leucomatous; anterior synechia	—	2/200	4.5 mm. Penetr.	3 yr. postop., L. P.	Clear; glaucoma
12 A. N.	M 50	L. E.	Scar	Dense	Many, small	20/200 without cor.	4.5 mm. Penetr.	2½ yr. postop., 3/200; no improvement w. gl.	Cloudy; infection developed postop.
13 A. No.	F 49	L. E.	Scars	Dense, central	Few	5/200 w. cor.	5 mm. Penetr.	6 mo. postop., w. −10.00 sph ⊃ −8.50 cyl, ax 155 = 20/60+3	Clear
		R. E.	Scars	Dense, central	Number hair-like; few deep	10/200 w. cor.; 8/200 without cor.	5 mm. Penetr., 5 mo. after op. on eye	1½ mo. postop., H. M.	Cloudy; vascularization and adhesions present
14 A. F.	M 77	L. E.	Opacity	All layers	—	H. M.	5 mm. Penetr.	2½ yr. postop., 2/200	Clear; poor vision due to glaucoma
15 P. H.	F 56	R. E.	Scar	Very dense, central	Few	3/200 without cor.	5 mm. Penetr.	3 yr. 9 mo. postop., w. −4.50 sph ⊃ −4.50 cyl, ax 105 = 20/60	Clear
16 L. A.	M 57	R. E.	Scarring following infection	Dense	Few, old	L. P.	5 mm. Penetr.	2 yr. postop., no improvement	Cloudy
17 A. D.	F 51	L. E.	Scarring following infection	Slight, diffuse	—	8/200 without cor.	6 mm. Penetr.	3 yr. 10 mo. postop., w. −10.00 sph = 10/200	Clear; semimyopic degeneration of fundus
18 V. B.	M 49	L. E.	Scarring following infection	Band-shaped, deeply pigmented, all layers	Few, hair-like, deep	10/200 without cor.; 20/200 w. cor.	6 mm. Penetr.	7 mo. postop., H. M.	Completely cloudy; vascularization in graft
		L. E.	Cloudy graft	Dense	At 2 o'clock	H. M.	6 mm. Penetr., 7 mo. after 1st op. in L. E.	1½ yr. postop., no improvement	Cloudy; some cosmetic improvement

* H. M., hand motion; L. P., light perception; penetr., penetrating; w. gl., with glasses.

In my series, 4 of 13 grafts followed two months or longer, or 30.8%, were clear (Table 2). Three of the clear grafts had had a follow-up of more than a year. Of the nine grafts listed as cloudy, one was three-fourths clear and one very slightly hazy. In an additional case the graft was clear one and a half months postoperatively.

In one of the patients in this series, a 57-year-old man with advanced Fuchs's dystrophy, the clouding of the cornea was such that the iris was not visible at the time of the first operation. The graft clouded over within a few weeks, but an excellent result was obtained on reoperation. The second graft has remained clear for well over a year. A good result was obtained in the other eye after one graft, although the patient's preoperative vision had been reduced to 5/200 (Case 1, Table 2).

It is my feeling that in Fuchs's dystrophy the graft should be done before the vision has been reduced to less than 20/200. Future findings will very likely demonstrate that the best results can be obtained in the very early stages of this disease, when there is not much visual impairment.

Other Findings.—The results in other diagnostic groups are of interest, although the numbers are too small for statistical significance. Of four cases of sclerosing keratitis followed two months or longer, clear grafts were obtained in two, with great visual improvement (Table 5). In band-shaped keratitis, a diagnosis termed unfavorable for keratoplasty by both Castroviejo[8] and Stansbury,[16] the results were somewhat poorer (Table 5). Four grafts were followed two months or longer, one being clear and three cloudy. An additional graft was clear one and a half months postoperatively. Five therapeutic grafts were done in patients with bullous keratitis. While none of these remained clear, nearly all the patients obtained symptomatic relief, although there was no visual improvement.

Therapeutic grafts were done in three cases of descemetocele, which Castroviejo classifies as less favorable for keratoplasty. Of these three grafts, which were followed up for more than half a year, one remained clear, while in the other two cases the operation made it possible to save the eyes. Three grafts done in cases of calcareous degeneration were followed for several years. One was clear at the end of eight years, while a second remained clear for more than five, clouding later. Of three cases with a diagnosis of scarring complicated by pigmentation, clear grafts were obtained in two. There were two patients with a diagnosis of pseudopterygium. One graft was clear five months postoperatively; the other was transparent one month after the keratoplasty. One graft was done in a 3-year-old patient with bilateral microphthalmos, secondary to old uveitis. While the graft was cloudy, the child's condition showed some improvement 15 months postoperatively.

Visual Results.—The aim in the majority of corneal grafts is to improve vision. Some, however, have a cosmetic, tectonic, or therapeutic purpose, no gain in vision being anticipated. Occasionally a graft done in the hope of better sight fails to improve the vision but achieves other benefits, i. e., subjective improvement or avoidance of enucleation. A transplant may be perfectly clear, but the visual acuity unchanged or reduced because of some unrelated condition, such as glaucoma or amblyopia.

In the present series, the visual result was tabulated in 222 of the 299 eyes in which the graft was followed two months or longer. Omitted from this analysis

TABLE 9.—*Results of Corneal Grafts in Chemical Burns, Explosion Injuries, and Other Traumatic Wounds**

Case No.: Patient	Sex; Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1† S. W.	M 46	R. E.	Sulfur dioxide burn	Dense	Few	L. P.	5 mm. Lamellar	10 mo. postop., no improvement	Graft cloudy; symblepharon improved; patient more comfortable
2 T. S.	M 25	L. E.	Sulfur dioxide burn	Deep and superficial	Fairly dense	H. M.	6 mm. Lamellar	7 mo. postop., no improvement	Moderate central haze; vascularization present; eye more comfortable
3‡ G. H.	M 41	L. E.	Old lime burn	Central, full thickness	Superficial	4/200 without cor.	6 mm. Lamellar	16 mo. postop., 6/200; no improvement w. gl.	Upper ⅓ of graft clear; lower ⅔ opaque; penetr. graft to be done
4 J. McD.	F 28	R. E.	lime burn	Nebulous scarring	Many fine	1/200 w. and without cor.	5 mm. Penetr.	.....	Eye enucleated 1 mo. after graft done
5 W. H.	M 36	R. E.	Old ammonia burn	Dense, white scarring	Superficial	1/200 without cor.	5.5 mm. Penetr.	1 yr. postop., no improvement	Partly cloudy; many blood vessels around graft
6 J. L.	M 27	R. E.	Ammonia burn	Dense scarring	Some	L. P.	6 mm. Penetr.	6 mo. postop., no improvement	Opaque; penetration of burn to iris and lens noted at operation; glaucoma present preop.
7 L. S.	M 40	L. E.	Acetic acid burns	Almost total	Several, superficial	L. P.	10 mm. Lamellar	1 yr. 10 mo. postop., H. M. at 3 ft.	Cloudy; dense scarring and many blood vessels present
8 W. K.	M 27	L. E.	Scarring (laboratory explosion); aphakia	Diffuse, many glass splinters present	Deep	2/200 without cor.; 5/200 w. cor.	6 mm. Lamellar	No follow-up	Clear; iris adhesions and cataract present; graft clear 3 mo. postop.
9 J. I.	M 38	R. E.	Old chemical burn	Dense, total	Many	H. M. at 2 ft.	6 mm. Penetr.	3 mo. postop., no improvement	Graft clouded
10 M. C.	F 40	L. E.	Scars from x-rays	Dense, central	Several	8/200 without cor.	5 mm. Penetr.	1½ mo. postop., no improvement	Cloudy
11 A. M.	M 27	R. E.	Traumatic scar (land mine explosion)	Very dense, total; sand and powder marks present	Some, fine, hair-like	20/200 without cor.; 20/100+ w. cor.	5 mm. Penetr.	6 mo. postop., no improvement	Clouded
12 J. K.	M 21	R. E.	Powder burn	Dense; powder embedded	—	H. M.	4.5 mm. Penetr.	2½ yr. postop., no improvement	Clear; traumatic cataract present
		L. E.	Powder burn	Total, moderate	Few, tiny	3/200	5 mm. Penetr., 10 mo. after op. on R. E.	1¾ yr. postop., no improvement	Clear; traumatic cataract present
13 J. M.	M 54	R. E.	Burn (hot lead explosion)	Complete	Very few	H. M. at 2 ft.	5 mm. Penetr.	3½ yr. postop., 20/200; no improvement w. gl.	Partly cloudy

		L. E.	Same as above	Very dense, central ¾	—	3/200	5 mm. Penetr., 3½ yr. after op. on R. E.	5 mo. postop., w. —6.00 sph = 20/50	Graft slightly hazy
14 T. R.	M 32	R. E.	Burn (explosion of molten metal); symblepharon	Dense leucoma	Slight	L. P.	5 mm. Lamellar	14 mo. postop., L. P.	Cloudy
15 J. Y.	M 32	L. E.	Multiple foreign bodies (dynamite explosion)	Diffuse	—	20/400 without cor.; 20/300 w. cor.	6 mm. Lamellar	6 mo. postop., w. cont. gl. 20/70	Clear
		R. E.	Multiple foreign bodies (dynamite explosion); aphakia	Diffuse	Minute, hair-like	2/200 without cor.; 20/200 w. cor.	6 mm. Lamellar	.....	.....
16 J. C.	M 29	L. E.	Scarring (dynamite explosion)	Myriad sand crystals and rust-stained spots	—	4/200 without cor.	6 mm. Lamellar	1 yr. 5 mo. postop., w. —2.50 sph ⌒ —3.25 cyl, ax 180 = 20/40	Clear
		R. E.	Same as L. E.	Same as L. E.	—	20/70 w. cor.	6 mm. Lamellar, 4 mo. after op. on L. E.	.....	Base of graft hazy; graft done preparatory to penetr. graft
		R. E.	Traumatic keratitis	Multiple foreign bodies around graft; base of graft hazy	—	20/200 without cor.	6 mm. Penetr., 2 mo. 3 wk. after 1st op. on R. E.	11 mo. postop., w. —7.00 sph ⌒ —4.00 cyl, ax 180 = 20/30	Clear
17 H. C.	M 36	R. E.	Scar (traumatic)	Leucoma, folds in Descemet's membrane	One, deep	H. M.	6 mm. Penetr.	3 mo. postop., w. +3.50 sph = 20/30	Clear
18 F. G.	F 33	L. E.	Scar (traumatic)	Dense, central	Many fine, in stroma	2/200	5 mm. Penetr.	.....	Recurrence of iridocyclitis; vascularization; eye enucleated 4 yr. postop.
19 F. V.	M 20	L. E.	Scar (perforating injury)	Very dense, central	Few	20/400 without cor.	5 mm. Penetr.	8 mo. postop., 15/200; no improvement w. gl.	Partly cloudy
20 H. J.	M 29	L. E.	Scar (traumatic)	Leucomatous	Large, superficial	L. P.	6 mm. Penetr.	No follow-up	Graft cloudy, with vascularization 3 mo. postop.
21 M. F.	M 38	L. E.	Scar (foreign body)	Central, one-half	—	4/200	5 mm. Penetr.	17 mo. postop., w. —9.00 sph ⌒ —4.00 cyl, ax 10 = 20/70	Clear

* Cont. gl., contact glass; cor., correction; gl., glasses; H. M., hand motion; L. P., light perception; penetr., penetrating; ph., pinhole; w., with.
† Operation done for therapeutic purposes.
‡ Not operated on by the author.

TABLE 10.—*Results of Corneal Grafts—Miscellaneous**

Case No.; Patient	Sex; Age, Yr.	Eye	Diagnosis	Corneal Opacity	Vessels	Preoperative Vision	Graft Size and Type	Postoperative Vision	Graft Clarity
1† A. G.	F 56	R. E.	Descemetocele (keratitis)	Dense, central; moderate, elsewhere	Slight	L. P.	5 mm. Penetr.	7 mo. postop., H. M. at 2 ft.	Cloudy; vascularization
2† S. P.	M 37	R. E.	Descemetocele (lime burn)	Very dense, pericentral	Number, above	M. M. at 2 ft.	5 mm. Penetr.	9 mo. postop., no L. P.	Cloudy
3† G. Y.	F 26	R. E.	Ruptured descemetocele (ulcer)	Central	Slight	M. M. at 1 ft.	6 mm. Penetr.	8 mo. postop., 20/50 w. cor.	Clear
4‡ W. T.	M 29	R. E.	Pseudopterygium (lime burn)	3 to 6 o'clock	Fairly large in pseudopterygium	20/50 w. cor.	Lamellar	1 mo. postop., w. +2.00 sph ⊃ −1.50 cyl, ax 120 = 20/25	Clear
5† K. K.	F 66	R. E.	Pseudopterygium, scarring	Limbal, superficial but dense	Few, nasally	5/200 without cor.; 20/200 w. cor.	6 mm. Lamellar	1½ mo. postop., 20/200; no improvement w. gl.	Clear, 5 mo. postop. (optic atrophy present preop.)
6 W.O'N	M 49	R. E.	Calcareous degeneration	Complete	Number	3/200 without cor.	5 mm. Penetr.	5½ yr. postop., 20/200; no improvement w. gl.; L. P.	Clear 5½ yr. postop.; cloudy, 11 yr. postop.; vascularization; calcareous degeneration
		L. E.	Calcareous degeneration	Complete	Number	L. P.	5 mm. Penetr., 7 wk. after op. in R. E.	3 yr. postop., no improvement	Cloudy
		L. E.	Cloudy graft; calcareous degeneration	Complete	Number	L. P.	5 mm. Penetr., 3 yr. after 1st op. in L. E.	6 yr. postop., no improvement	Cloudy; vascularization; calcareous degeneration; secondary glaucoma
7 A. Z.	F 70	R. E.	Calcareous degeneration	Dense, total	Moderate, superficial	H. M.	4.5 mm. Penetr.	8 yr. postop., L. P.	Clear; complications from cataract extraction
8 M. A.	F 35	R. E.	Old scar complicated by pigmentation, resulting from chemical treatment	All layers	—	20/50 without cor.; 20/30 w. cor.	4.5 mm. Penetr.	7 mo. postop., w. −14.50 sph ⊃ −4.50 cyl, ax 150 = 20/40—1	Clear

9 A. B.	F 70	L. E.	Old keratitis, pigmentation in superficial layers, resulting from tattooing	Deep opacities throughout	Minimal, peripheral	2/200 w. and without cor.	6 mm. Penetr.	13 mo. postop., w. +4.50 sph ⊃ +3.50 cyl, ax 80 = 20/70+	Clear
		R. E.	Same as in L. E.	Same as in L. E.	Peripheral	2/200 without cor.	6 mm. Lamellar, 4 mo. after op. in L. E.	5 mo. postop., C. F.	Cloudy; recurrence of keratitis; immature cataract; penetr. graft to be done
10 L. S.	F 3	L. E.	Bilateral microphthalmos, secondary to old uveitis	Dense	Many, large and small	L. P. (?)	5 mm. Lamellar	15 mo. postop., a little improvement	Some clouding
11 J. D.	M 46	L. E.	Scarring, following keratouveitis	Macula	Few, marginal	5/200 without cor.; 10/200 w. cor.	6 mm. Penetr.	No follow-up	Graft translucent 4 mo. postop.; iris adhesion above (cataract and chorioretinal degeneration present preop.)
12‡ D. P.	F 29	R. E.	Birth injury	Hazy, thickened, edematous; superficial bullae	—	L. P.	6 mm. Penetr.	3 mo. postop., w. −4.00 sph ⊃ +8.00 cyl, ax 90 = 20/400	Cloudy; graft done for relief of discomfort

* Penetr., penetrating; L. P., light perception; H. M., hand movement; C. F., counts fingers; w. gl., with glasses.

† Operation done for therapeutic purposes.

‡ Not operated on by the author.

were therapeutic grafts not followed by visual improvement and cases in which glaucoma, amblyopsia ex anopsia, cataract, or degenerative disease of the posterior segment was present and there was no visual gain. Four therapeutic grafts were included in the tabulation because of substantial improvement in vision and, in addition, a small number of cases in which great visual gain occurred despite the presence of other ocular disease. This made a total of 222 cases followed up for two months or longer in which the final visual result could be assessed. When two or more operations were done on the same eye, the visual result following the last graft was compared with the vision before the first graft was performed. Small improvements in vision were counted only in the presence of a clear graft.

Of these 222 cases, 160, or 72.1%, showed visual improvement. Fifty-seven, or 25.7%, had a final vision of 20/30 or 20/20. Forty-six, or 20.7%, had the same vision before operation, while 13, or 5.9%, had a reduction in vision. In three

TABLE 11.—*Relationship of Diagnosis to Percentage of Clear Grafts in Major Diagnostic Groups*

	Present Series		Owens and Associates [9]		Roberts [12]	
Diagnosis	No. Cases	Clear Grafts Followed 2 Mo. or More	No. Cases	Clear Grafts Followed 2 Mo. or More	No. Cases	Clear Grafts Followed 4 Mo. or More
Keratoconus	84	89.3%	63	65.2%	20	85%
Hereditary dystrophy	28	85.7	17	58.8	8	100
Nonspecific scarring	16	68.8	141	46.9	42	42.8
Scarring, inactive ulcer	18	66.7	..	....	..	..
Interstitial keratitis	45	62.2	49*	49.0	12*	50
Leucoma	9	55.6	..	....	..	..
Active ulcer	6	50	13†	23.1	1‡	0
Trachomatous scarring	11	45.5	..	....	3	2
Burns, explosions, trauma	23	39.1	..	....	..	..
Chemical burns	8	0	24	20.8	6	0
Explosion injuries	10	70	..	....	..	..
Traumatic injuries	5	40	22	18.2	8	50
Adherent leucoma	17	35.3	..	....	..	..
Fuchs's dystrophy	13	30.8	10	0	3	0

* Scar of syphilitic interstitial keratitis.
† Active keratitis or ulcer.
‡ Mooren's ulcer.

cases, or 1.35%, enucleations were done within one and a half years after the operation because of the development of panophthalmitis.

As a further gauge of visual gain, a tabulation was made of the cases in which the vision improved from 20/200 or less before operation to 20/100 or better, as was done in the study of Owens and associates.[9] This improvement was made in 114 cases out of 185 with preoperative vision of 20/200 or less, or 61.6%.

A check was also made to evaluate the visual results of keratoplasty in patients with a preoperative vision of 20/100 or better. Thirty-four such patients were operated on. Twenty-six, or 76.5%, showed postoperative visual improvement; 4, or 11.76%, had the same or almost the same vision, and 4, or 11.76%, had poorer vision.

Results in Cases of Multiple Grafts.—Published reports differ as to the feasibility of trying a second, or even a third, corneal graft after earlier failure. Franceschetti and Bischler [8] report success with secondary operations, while Stansbury [15] found the results poor in his series. In my series, a special tabulation was made of the postoperative vision and graft clarity in five cases in which three

successive corneal grafts were done on the same eye. In three of these, the graft was clear more than two months after operation. Comparison of the vision prior to the first graft and following the last showed visual gain in two cases; in the third, the vision was reduced, anterior synechias and a cataract being present. In a fourth case, the graft was fairly clear following the third operation, but there was no visual improvement. The fifth patient had a cloudy graft for the final result, with no gain in vision.

Lamellar Grafts.—Views differ on the newer technique of the lamellar graft.¶ It gave successful results in 75% of 270 operations by Paufique.# He advocates its use in superficial opacities, the hereditary dystrophies, interstitial keratitis, in nervous patients, persons with only one eye, and those with aphakia or nystagmus, among others. Castroviejo [5] reports the results of this technique in 39 cases, 20% of which showed visual gain. He concludes that, while the technique is safer than the penetrating graft, avoiding such unpleasant complications as iris incarceration and prolapse, it gives less brilliant results.

A total of 54 lamellar grafts were included in my series. Of these, 12 were performed for therapeutic purposes, 10 having been followed up for more than two months. Of these 10, 8 were considered successful, the criterion being relief of symptoms. One additional graft was clear one month postoperatively. Four lamellar grafts were done in preparation for a penetrating transplant. One of these was clear two months postoperatively; the other three clouded, with no improvement in vision. Thirty-five of the remaining 38 lamellar grafts were followed for two months or more. Fifteen of these were clear, while 19 were cloudy; one graft was lost as the result of too early cortisone therapy. Two grafts were clear on discharge, with no follow-up, while one was translucent three weeks postoperatively.

SUMMARY AND CONCLUSIONS

The results of 365 corneal transplantations in 330 eyes have been analyzed statistically.

A total of 299 cases were followed up for two months or longer, 193 grafts, or 64.5%, remaining clear.

The percentages of clear grafts in the various diagnostic groups were tabulated, as follows: keratoconus, 89.3%; Groenouw's dystrophy, 85.7%; nonspecific scarring, 68.8%; scarring from inactive ulcer, 66.7%; interstitial keratitis, 62.2%; leucoma, 55.6%; active ulcer, 50%; trachomatous scarring, 45.5%; chemical burns, explosion injuries, and traumatic wounds, 39.1%; adherent leucoma, 35.3%; Fuchs's dystrophy, 30.8%.

Not all corneal graft cases lend themselves to exact prognostic classification. In all cases of scarring, the operative result depends on the extent of preoperative opacity, the degree of vascularization, and the time interval between the subsidence of the active inflammatory process and the operation.

It is my experience that a definite prognosis as to the operative result can be made in a high percentage of cases and that many useless operations can thus be avoided.

¶ References 5, 10, and 11. DeVoe, in Paufique.[10] Paufique, cited by Castroviejo.[5]

Cited by Castroviejo.[5]

The visual result was tabulated in 222 of the 299 eyes in which the graft was followed two months or longer. Of these, 160, or 72.1%, showed visual improvement; 46, or 20.7%, had the same vision as before operation, and 13, or 5.9%, had a reduction in vision. There were three enucleations within one and a half years after the keratoplasty, and two others, four years after operation.

Of 185 cases with a preoperative vision of 20/200 or less, 114, or 61.6%, improved to 20/100 or better after the transplant.

In 34 cases the preoperative vision was 20/100 or better. Twenty-six, or 76.5%, had better vision postoperatively; 4 had the same vision, and 4 had poorer vision.

Fifty-four of the 365 grafts in the series were lamellar grafts. A brief summary of the results in these transplants is given.

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EFFECT OF VASODILATOR DRUGS AND STELLATE GANGLION BLOCK UPON THE ELECTRORETINOGRAM

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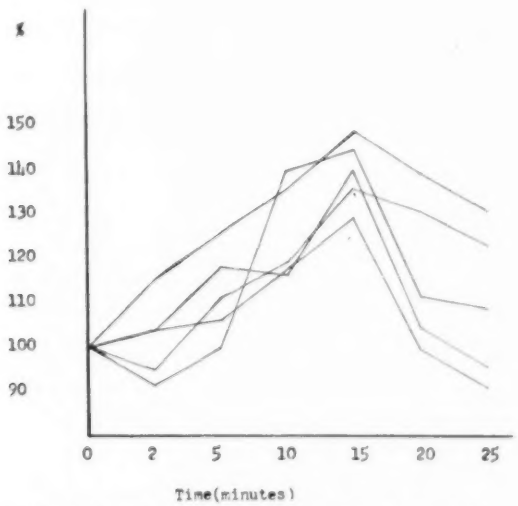
THE ELECTRICAL potential developed in the human retina upon stimulation by light is detected and recorded, by the technique described by Karpe,[1] as the electroretinogram (ERG). The amplitude of this potential has been shown, by Henkes [2] (1951), to increase upon the systemic administration of vasodilator drugs.

With the knowledge that vasodilators cause an increase in the amplitude of the b-wave of the ERG, it was thought that the electroretinographic technique might



Graph 1.—Glyceryl trinitrate, 1.2 mg. sublingually.

allow for graphic comparison of the relative efficacy of several vasodilators upon retinal blood flow. Accordingly, four common vasodilating drugs were selected and studied. In addition, the effect of a unilateral stellate ganglion block using 1%

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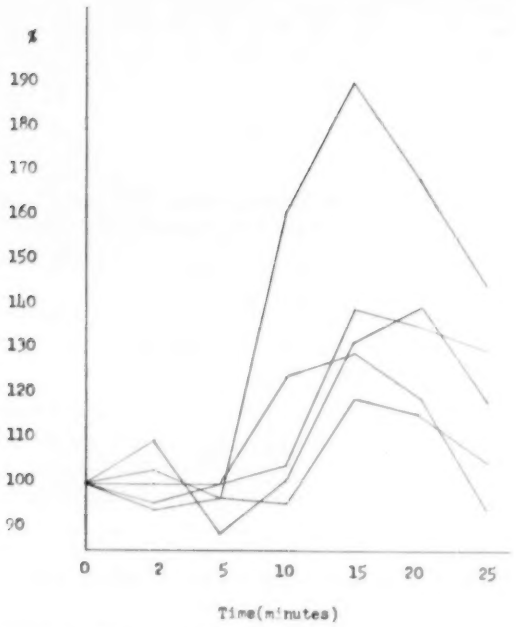
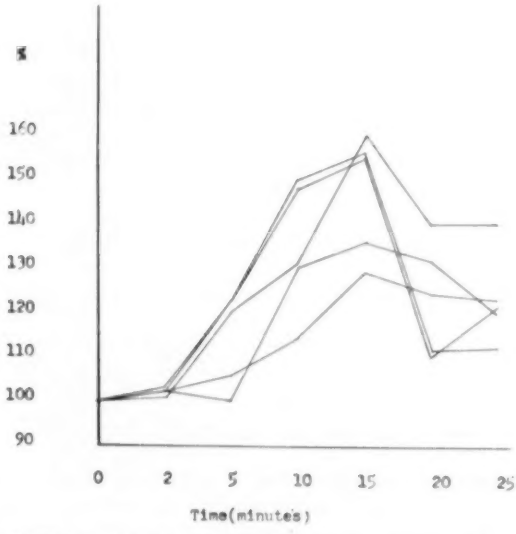


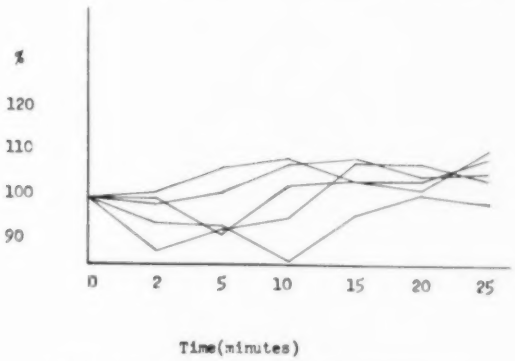
Graph 2.—Papaverine hydrochloride, 120 mg. intravenously.



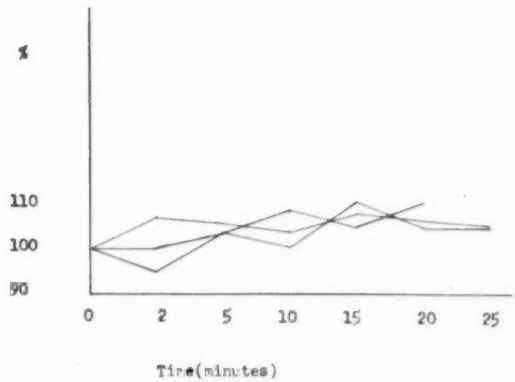
Graph 3.—Tolazoline (Priscoline), 50 mg. intravenously.

lidocaine (Xylocaine) hydrochloride was determined. The stellate ganglion block was performed on the same side as that used for measuring the ERG's. Serial ERG's were taken before and after the administration of the drugs and block.

The four drugs used and their dosages were as follows: glyceryl trinitrate (nitroglycerin), 1.2 mg. sublingually; tolazoline (Priscoline*), 50 mg. intravenously; papaverine, 120 mg. intravenously, and nicotinic acid, 100 mg. intravenously. Five normal 18- to 20-year-old volunteer men were subjects for each drug, and three similar subjects for the ganglion block.



Graph 4.—Nicotinic acid, 100 mg. intravenously.



Graph 5.—Stellate ganglion block (lidocaine).

The procedure was as follows: After 10 minutes of dark adaptation a series of ERG's was recorded. The drug or block was then administered, without disturbing adaptation. The stellate block was not considered to be present unless definite anhydrosis, relative miosis, and some degree of ptosis were noted. ERG's were recorded at 2, 5, 10, 15, 20, and 25 minutes following block or administration of the dose. The resultant ERG's were measured with a vernier caliper and the values calculated in terms of the base line b-wave, this base line value being considered as 100%.

It is our opinion that an error of at least 10% is inherent in this method. Personal contact with Henkes has revealed that this method is similar to the technique he employs. The results are tabulated (Tables 1 to 6) and presented graphically (Graphs 1 to 5).

* The Priscoline used in this study was supplied by Ciba Pharmaceutical Products, Inc.

TABLE 1.—*Increments* in ERG Amplitude with Glyceryl Trinitrate, 1.2 mg. Sublingually*

Time, Min.	Subjects				
	1	2	3	4	5
0	100	100	100	100	100
2	92	95	115	108	103
5	100	112	126	106	118
10	140	118	136	117	116
15	145	136	148	129	141
20	112	131	139	99	104
25	108	123	131	92	96

* Values in terms of the base line b-wave.

TABLE 2.—*Increments in ERG Amplitude with Papaverine Hydrochloride, 120 Mg. Intravenously*

Time, Min.	Subjects				
	1	2	3	4	5
0	100	100	100	100	100
2	94	110	103	100	96
5	97	88	97	100	100
10	162	101	96	123	104
15	191	132	119	129	139
20	169	140	116	119	136
25	145	118	105	94	130

TABLE 3.—*Increments in ERG Amplitude with Tolazoline, 50 Mg. Intravenously*

Time, Min.	Subjects				
	1	2	3	4	5
0	100	100	100	100	100
2	103	102	101	102	102
5	123	106	120	100	123
10	148	114	131	131	150
15	155	129	160	136	156
20	110	124	140	132	111
25	122	123	140	120	112

TABLE 4.—*Increments in ERG Amplitude with Nicotinic Acid, 100 Mg. Intravenously*

Time, Min.	Subjects				
	1	2	3	4	5
0	100	100	100	100	100
2	94.2	100	98.7	101	88
5	94	92.8	101	107	93
10	86	103	108	109	96
15	96	104	109	104	108
20	101	102	105	104	107
25	99	111	106	109	104

TABLE 5.—*Increments in ERG Amplitude with Stellate Ganglion Block (Lidocaine)*

Time, Min.	Subjects		
	1	2	3
0	100	100	100
2	107	100	95
5	106	104	104
10	104	101	108
15	108	111	105
20	107	105	111
25	106	105	...

RESULTS

It is our interpretation that these results indicate no significant increase in the amplitude of the ERG with nicotinic acid or stellate ganglion block, despite the intense flush and subjective feeling of warmth noted by the patients after administration of nicotinic acid.

Each of the three other drugs (glyceryl trinitrate, tolazoline, and papaverine) was followed by an increase, with the greatest increase 15 minutes after administration in all cases but one. In this instance the increment was greatest after 20 minutes. In each case the ERG was returning toward the original level by 25 minutes.

These data do not allow for any conclusion as to the most effective drug, since there was no uniformity in the degree of reaction to the several drugs. Three subjects were given each of the drugs that caused an increase in ERG amplitude. A summary of the maximum increment in each of these subjects with the three drugs is presented in Table 6. It is seen that Subject 1 reacted most highly to papaverine,

TABLE 6.—*Comparison of Maximum Increment*

Drug	Subjects		
	1	2	3
Papaverine	191	140	119
Tolazoline	155	129	160
Glyceryl trinitrate	145	136	148

and Subject 3 to tolazoline, and that the responses of Subject 2 were of equal degree, within the limit of error of the method, to all three drugs.

COMMENT

The amplitude of the ERG has been shown to increase under the influence of certain vasodilator drugs. Whether this effect is the result of an increased retinal arterial blood flow or is due to added blood supply by the choroidal vessels is open to some question.

The b-wave of the ERG is thought, by the authorities in the field, to arise in the synaptic layers of the retina, probably in the bipolar cells. The deep net of the retinal arterial capillaries extends down to the outer border of the inner nuclear layers, where these cells are situated. These retinal capillaries are thought to be the main blood supply of these cells.

The rods, however, which initiate the chain of responses resulting in the activity of the bipolar cells, are supplied with nutrients by the choriocapillaris. Any change in the level of function of the rods, which might result from fluctuation in the choroidal blood flow, might be expected to influence bipolar cell activity and hence the ERG.

Leopold [3] has shown, by direct observation of exposed choroidal vessels, that neither superior cervical sympathetic ganglionectomy nor nicotinic acid affect the caliber of the outer layers of choroidal blood vessels, although he could not study the choriocapillaris.

It is of interest to note that the results obtained from the four drugs studied and from stellate ganglion block are comparable to results reported by Scheinberg and Jayne [4] on the effect of some of these drugs of stellate ganglion block on cerebral blood flow. These studies, using the nitrous oxide technique of Kety and Schmidt,

showed no increase in cerebral blood flow with stellate ganglion block or nicotinic acid. Papaverine did increase cerebral blood flow.

Since stellate ganglion block produced no marked change in retinal blood flow, we feel that this is an indication that the sympathetic nervous system plays a minor role, if any, in the control of normal retinal blood flow. However, since these studies were carried out on normal young men, the results obtained do not preclude the possibility that stellate ganglion block may under certain abnormal conditions, such as retinal vasospasm, increase retinal blood flow.

CONCLUSIONS

Neither sympathetic block nor intravenous administration of nicotinic acid causes an increase in the amplitude of the ERG, while tolazoline, papaverine, and glyceryl trinitrate each causes an increment of measurable degree in the height of the b-wave of the ERG.

The maximum effect is present at 15 minutes after administration of the drug. It is inferred that the last-mentioned three drugs are more effective than nicotinic acid or stellate ganglion block in increasing retinal blood flow.

Individual differences in the reactions of different subjects to the drugs preclude accurate determination of the most effective of these drugs by this means, at least in young normal men.

The absence of any marked effect of sympathetic block is felt to indicate that the sympathetic nervous system has little control over that blood flow which affects the amplitude of the ERG in normal eyes.

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MALIGNANT MELANOMA OF THE CORNEA

Report of a Case

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RARELY, the cornea may be the site of a primary malignant melanoma. In such instances the tumor arises from the Schwannian cells of the corneal nerves or in the basal cells of the corneal epithelium. Reese [1] states that the basal cells of the corneal epithelium are potential melanoblasts, and this is evidenced by the melanin which these cells show in Stähli pigmented lines, in Hudson lines, and as an occasional sequela of irradiation. Reese [1] published a photograph of a malignant melanoma of the cornea in an 89-year-old man who had noted a progressively enlarging lesion for two years. Serial sections in this case showed no connection with the limbus.

The cornea is more commonly invaded by a malignant melanoma which has arisen in the region of the limbus, this invasion usually taking place between the epithelium and Bowman's membrane, with subsequent invasion of the substantia propria.

REPORT OF CASE

F. C., a white man, aged 53, complained of several attacks of severe pain in the left eye over a period of two years. During this time there was also gradual loss of vision in the eye, but he did not seek treatment until three days before he was admitted to the hospital for enucleation. When 12 years of age, he had sustained an injury to this eye, but any details as to its severity was unobtainable.

Ophthalmoscopic Examination.—Left eye: Visual acuity, counting fingers at 1 ft. (30 cm.). The eye was extremely painful, showing marked pericorneal injection and increased intraocular tension. Right eye: Vision normal; external eye and fundus normal.

The left eyeball was enucleated and submitted to the pathology department of the Kresge Eye Institute for histological examination.

Gross Examination.—The eye measured 22 mm. anteroposteriorly, 24 mm. in its horizontal diameter, and 24 mm. in its vertical diameter. The cornea measured 11 mm. in both the horizontal and the vertical meridian. A tumor mass was noted on the cornea (Fig. 1); the tumor was pigmented, was oval in shape in the horizontal meridian, measured 8 mm. horizontally and 5 mm. vertically, and was elevated about 2 mm. Its apex was ulcerated. The tumor was located in the lower portion of the cornea, leaving considerable clear cornea above but with a clear line of demarcation between it and the limbus about its entire periphery.

Histological Examination.—The tumor was made up of spindle-shaped cells arranged in sheets and whorls showing moderate pigmentation and vascularization (Fig. 2). Special stains reveal marked reticulum content. This mass reaches its greatest thickness near the center of

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the cornea and thins out as the region of the limbus is approached. The tumor is situated between the epithelium and Bowman's membrane. Bowman's membrane is intact throughout, except for a few areas where the substantia propria has been very slightly invaded superficially. The substantia propria shows no evidence of deep infiltration. There is no evidence of Schwann cell proliferation. The epithelium on one side of the tumor presents an intact basement membrane (Fig. 3). The epithelium on the other side of the tumor shows massive proliferation of the basal epithelial cells, this activity extending in several sections to the region of the limbus, without actual elevation into tumor formation (Fig. 4). The epithelium is absent over the apex

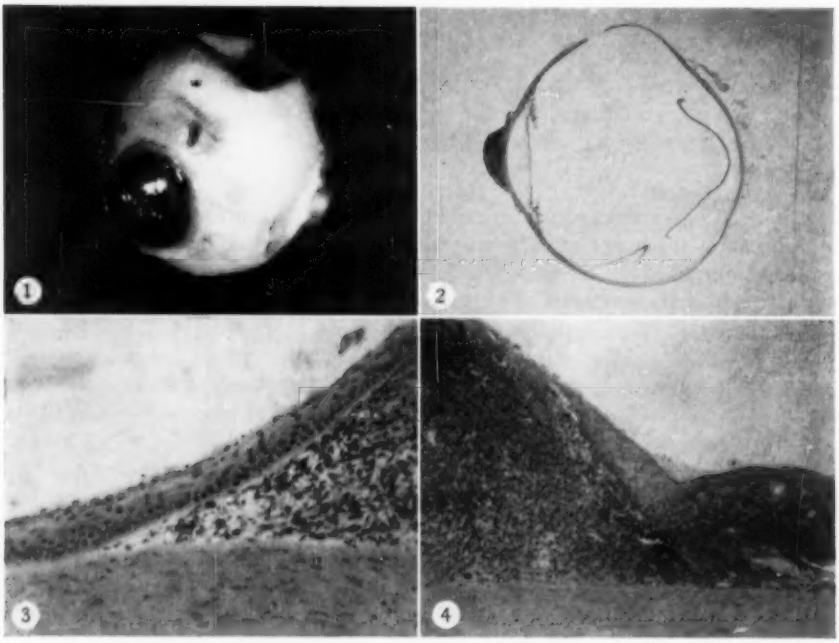

Fig. 1.—Tumor mass on cornea. The mass is elevated about 2 mm. at its apex and does not reach the limbus.

Fig. 2.—Malignant melanoma of the cornea.

Fig. 3.—Basal cells of the corneal epithelium showing no proliferation or downward growth.

Fig. 4.—Basal cells of the corneal epithelium showing marked proliferation and downward growth.

of the tumor, owing to trauma. All sections were reviewed from limbus to limbus, with no evidence of primary extension from the limbus or of extension to or from the substantia propria.

Diagnosis: Malignant melanoma of the cornea.

SUMMARY

This case is presented as one of malignant melanoma of the cornea arising from the basal cells of the corneal epithelium. The activity of these basal cells is noted particularly on one side of the tumor, where it extends laterally toward the limbus.

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CONGENITAL ALACRIMA IN FAMILIAL AUTONOMIC DYSFUNCTION

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ALTHOUGH congenital absence of tears is a rare defect, its existence has been known since Thurnam's [1] report of two cases in 1848. Under the title of alacrimia congenita Sjögren and Eriksen,[2] in 1950, discussed this subject, and in 1952 Sjögren [3] reviewed 14 of the cases reported in the ophthalmic literature without establishing a common etiologic factor. Among the ascribed causes were (1) persistence of the physiologic condition of the newborn, (2) hypoplasia or absence of the lacrimal gland, and (3) disturbance of innervation of the lacrimal gland. In addition to the previously reported cases, there is a condition in which congenital alacrima is a constant finding. Credit for the recognition of this clinical entity goes to Riley [4] and his co-workers, who in 1949 reported five such cases under the title of "Central Autonomic Dysfunction with Defective Lacrimation." Largely as the result of subsequent studies by Riley,[5] who collected 33 such cases, a definitive clinical picture has emerged which he states is characterized by "evidence of vegetative dysfunction, such as excessive perspiration, drooling, erythematous blotching of the skin, intermittent hypertension and defective lacrimation, along with hyporeflexia and emotional instability." Since the ophthalmic literature contains only brief references * to this disease, a more detailed description will be presented.

GENERAL CONSIDERATIONS

This condition, which to date has been described only in children of Jewish extraction, has occurred in siblings in approximately one-third of the reported cases. Both sexes are involved with equal frequency.

The initial finding is an absence of tears in an infant who has difficulty in swallowing and presents a genuine feeding problem. Other symptoms appear as the child gets older, and the diagnosis can usually be made at the age of 2 years. The child then drools constantly, is undernourished, is extremely awkward, and walks on its toes with a shuffling gait. Violent emotional outbursts, which occur frequently and without provocation, make the management of such a child a real problem.

Etymologically "alacrima" seems preferable to alacrimia.

Read at the meeting of the American Ophthalmological Society at Glacier National Park, Mont., June 16, 1954.

From the Institute of Ophthalmology of Presbyterian Hospital and Columbia University College of Physicians and Surgeons.

* References 6, 7, and 8.

Foremost among the associated systemic conditions are cyclic vomiting and paroxysmal hypertension. The attacks of vomiting may last a few days or several months. During these episodes severe personality changes often take place. The child becomes sullen, uncommunicative, and incorrigible. There is a concurrent rise in blood pressure, which may or may not be severe. In some instances this hypertension has so paralleled that found in pheochromocytoma that surgical exploration has been undertaken. When such a child is upset, not only may the blood pressure rise to undue heights, but excessive perspiration occurs and sharply defined blotches appear over the upper part of the body (Fig. 1). These signs usually disappear in 10 to 15 minutes after the source of excitement is removed. In many of these children, despite normal tactile sense, there is a marked indifference to pain. In some instances bruises, and even fractures, have gone unnoticed. Repeated

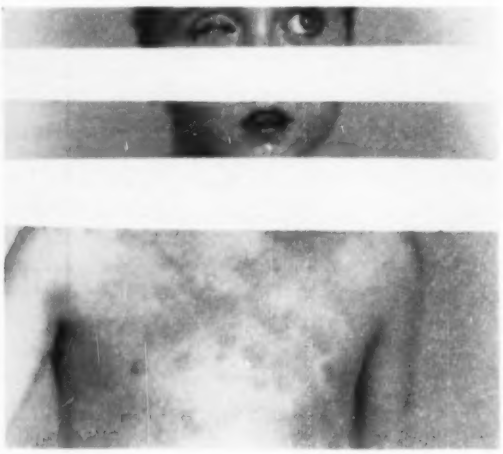

Fig. 1 (Case 1).—Familial autonomic dysfunction, showing blotching of skin and drooling.

attacks of bronchopneumonia are a frequent complication. Eight of the 10 deaths reported in Riley's series were attributed to aspiration pneumonia.[5] These children also are subject to high fevers of unexplained origin. The "all or none" response these patients make to minor emotional stimuli probably was responsible for the early erroneous impression that mental retardation invariably accompanied this disorder. It is now known that in some instances the intelligence may even be above normal. While convulsions do occur in about one-half of the cases, they are of no diagnostic significance. Breath holding and urinary frequency are also common incidental findings.

OCULAR MANIFESTATIONS

The two ocular findings which are constantly present in this disease are (1) defective lacrimation and (2) corneal hypesthesia. The child's failure to secrete tears when crying is usually the first abnormality noted by the parents. This alacrima does not produce a dry eye, nor is there any evidence of lack of conjunctival secretion. Although the eyes appear normal, no more than a drop or two of tears

is secreted. Lacrimation cannot be induced by external irritation or stellate ganglion block, and the Schirmer test shows an almost complete absence of tear secretion.

In these patients evaluation of the corneal sensitivity is difficult because of their relative indifference to pain, but it appears to be either completely absent or greatly diminished in all of them. Ample clinical evidence supports this contention, for corneal lesions, e. g., abrasions and foreign bodies, are productive of little, if any, discomfort. In view of this apparent corneal hypesthesia, it is interesting to note that in two-thirds of the cases recorded by Riley the corneas had not become involved.[5] Even so, corneal ulceration does occur and may reach serious proportions. Riley reports that in 3 of the 11 cases with corneal lesions the sight became seriously and permanently impaired. Usually the presence of these ulcers is first detected by the parents, who notice a white spot on the cornea. At this time the eye is only slightly injected; photophobia and lacrimation are absent, and the child has no appreciable discomfort (Fig. 2). The superficial indolent ulcer with a

Fig. 2 (Case 3).—Familial autonomic dysfunction, showing typical corneal ulcer scar with absence of inflammatory reaction.

central slough tends to spread and deepen until it perforates. Bacteriological studies are unrevealing, being either negative or demonstrating only the usual secondary invaders.

TREATMENT

The treatment of this condition has been directed largely toward relief of its most distressing symptoms. Therefore, various and sundry remedies have been tried, among which are anticonvulsants, sedatives, muscle relaxants, corticotropin, and psychotherapy, and even lumbar sympathectomy in one case with arterial hypertension.[5] None of these measures has been of lasting benefit. The corneal ulcers have also received many forms of therapy, including curettage, with and without cauterization, and local and systemic administration of various antibiotics and vitamins. The results with these methods were most discouraging, but as the clinical picture has become more clearly defined, a different therapy has been reasonably successful. Protection of the cornea by creating two intermarginal lid adhesions has become the standard treatment. This permanent closure is used even in the presence of a dirty, sloughing ulcer. With this treatment, the ulcer heals promptly,

the infiltration becomes less pronounced, and the cornea clears to a remarkable extent. This state of affairs persists as long as the lids are closed, but the corneal ulceration may recur when the eye is opened. To reduce the frequency of this complication, it is recommended that the lid adhesions be left for at least six months. If, as is the usual practice, the lids are closed with two intermarginal adhesions, it is wise to sever one and wait a few weeks more before cutting the other one. In this way one can better judge the resultant effect on the cornea.

REPORT OF CASES

CASE 1.—H. R.,† a 19-month-old Jewish boy, was first seen in Vanderbilt Clinic on Jan. 31, 1945, because of a severe corneal ulcer, left eye, of six months' duration. Conservative treatment with eye drops and ointments had been unsuccessful.

Past History.—The boy's mother stated that the child had always presented a feeding problem and that he had been unable to sit alone until he was 15 months of age.

Physical Examination.—Although the child was somewhat small for his age, it was felt that his physical development was within normal limits. He appeared to have no discomfort from his eye lesion, drooled constantly, and showed severe motor incoordination. He was unable to stand unsupported, and speech was at about the 10-month level. Psychometric examinations showed marked mental retardation.

Since the second and third years of life the patient has shown extreme emotional instability, blotching of the skin, and excessive perspiration. There has been absence of deep tendon reflexes, and he walks on his toes with a shuffling gait. The blood pressure has been very labile, but no genuine hypertension has been present.

In the past eight years this patient has had 12 hospital admissions for repeated attacks of bronchopneumonia or episodes of cyclic vomiting.

In 1948, diencephalic compression at the tentorial level was thought to be a factor because of the lack of tearing associated with intermittent outbursts of autonomic overactivity. Therefore, division of the tentorium was performed, and for a few days drooling, blotching, and excessive perspiration disappeared and it was thought that the eyes appeared more moist. However, within two weeks all signs had returned.

Ocular Examination.—At the time of the first examination the right eye appeared to be normal. The lids of the left eye were normal, and the conjunctival injection was only slight. A deep ulcer involved the central two-thirds of the cornea. There was no tearing in either eye, and on several occasions it was shown that litmus paper was not moistened beyond the lid margins. Corneal sensitivity was very much reduced in both eyes. No weakness of the ocular muscles was observed. The fundi were not seen.

Treatment.—Despite iontophoresis with penicillin, the ulcer became worse, and within a few weeks the cornea perforated and a conjunctival flap was drawn over this area. Since then there has been a large, dense, vascularized central corneal opacity. Recently a superficial keratectomy with lid adhesions was performed on the left eye. The result of this surgery has not yet been determined.

In March, 1945, about six weeks after the initial examination, slight conjunctival injection and a central corneal ulcer were observed in the right eye. Because of the feeding problem, a tentative diagnosis of keratomalacia was made and intensive vitamin therapy combined with various antibiotics used, but the corneal ulcer did not heal. In October, 1945, intermarginal adhesions were placed between the lids of the right eye. During the ensuing years, the eye has remained quiet when closed, but the corneal ulcer has recurred each time the adhesions are severed. There is at present a central corneal opacity with some deep vascularization at the lower limbus. The vision is now 15/200 in this eye.

CASE 2.—R. L., a 4-year-old Jewish boy, was first seen on Sept. 14, 1948, because of a recurrent corneal ulcer, left eye, of one year's duration. Treatment elsewhere, consisting of the intensive use of antibiotics both systematically and locally, vitamin therapy, and chemical cauterization of the ulcer had not been helpful.

† Reported by Riley, Day, Greeley, and Langford.[4]

Past History.—The parents stated that the patient had feeding difficulties in infancy but that this problem no longer existed. Because of the presence of bilateral corneal hypesthesia, the possibility of a brain tumor with secondary neuroparalytic keratitis was considered. Extensive studies revealed no evidence of any intracranial process.

Physical Examination.—At the time of the first examination the patient showed marked drooling, difficulty in walking, and some emotional instability. Deep tendon reflexes were absent, and the blood pressure was very labile. During the intervening six years, blotching of the skin and cyclic vomiting have appeared. Marked lability of the blood pressure persists, but no true hypertension has developed.

Ocular Examination.—There was bilateral absence of tears and reduced corneal sensitivity. The Schirmer test showed 1 mm. of wetting after five minutes. In the left eye there was a central corneal ulcer 3 mm. in width and surrounded by a zone of edema and infiltration 3 to 4 mm. wide. Deep vascularization was also present near the limbus. The right cornea was clear; the pupils were equal and active, and the extraocular muscle balance and fundi were normal. A tentative diagnosis of neuroparalytic keratitis was made, but after further investigation of the patient's general condition it was changed to familial autonomic dysfunction.

Treatment.—Intermarginal adhesions were placed in the left eye. One year later these adhesions were cut. Since that time the cornea has remained healed, although a diffuse opacity persists.

CASE 3.—G. L., a 6-year-old Jewish boy, was first seen on May 18, 1953, because of a recurrent corneal ulcer, right eye, of three months' duration. Antibiotics and cortisone had not influenced the healing of this lesion.

Past History.—The parents stated that this child had never cried and had had difficulty in swallowing since birth. He had also had many attacks of bronchopneumonia and fevers of unknown etiology. He was easily upset and on such occasions developed patchy flushes and excessive perspiration. His parents noticed that he carried his left arm in an unusual position. X-rays showed an old fracture of the left humerus, which apparently had caused no discomfort.

Physical Examination.—This boy was poorly developed and walked on his toes with a shuffling gait. He was drooling and had a nasal type of speech. There were dystonic movements of the arms and legs and absence of deep tendon reflexes. The initial blood pressure reading was 152/120 mm. Hg. but became less when the child adjusted to his surroundings.

Ocular Examination.—The lids and conjunctiva of both eyes appeared normal. There was bilateral absence of tears, along with diminished corneal sensitivity. In the Schirmer test the litmus paper was not moistened beyond the lid margins, nor was any tearing demonstrable after a bilateral stellate ganglion block. A large shallow ulcer with some deep vascularization was present in the right cornea. The left cornea was clear. The pupils were of equal size and reacted normally. The extraocular muscles were in good balance, and the fundi were normal.

Treatment.—Intermarginal adhesions were placed between the lids of the right eye. After this treatment the ulcer healed rapidly. Six months later, when the lid adhesions were cut, a diffuse, nonstaining corneal opacity was observed. During the ensuing four months the cornea has remained healed. A tear substitute, consisting primarily of methylcellulose, is being instilled in each eye at frequent intervals.

On a biopsy specimen of the right lacrimal gland, taken at the time of severing the intermarginal adhesions, Dr. Joseph A. C. Wadsworth has submitted the following report: "The specimen is composed of what appears to be normal lacrimal gland. There are some localized areas of lymphocytic infiltration and areas of fibrosis. These changes are within the range of normal, but one gets the impression that there is a greater amount of connective tissue than is usually seen in the normal lacrimal gland."

COMMENT

Children with this congenital alacrima are seen by oculists either because they do not cry or because they have an indolent corneal ulcer. To establish a diagnosis, we must consider those conditions found in children in which there may be a deficiency of tearing, sometimes associated with corneal changes.

Congenital Alacrima.—In 1848, Thurnam [1] described two middle-aged cousins who showed, in addition to complete absence of tears, a lack of scalp hair, eyebrows and eyelashes, a prominent brow, and severe defects in the development of teeth. They had never perspired and were very sensitive to warm weather. No corneal changes were reported, nor was there mention of any disturbance in the corneal sensitivity. In 1929, Weech [9] classified Thurnam's cases as belonging to a group called anhidrotic hereditary ectodermal dysplasia.

Since this original publication at least 18 cases of congenital alacrima have been reported.‡ The systemic changes associated with this deficiency differ greatly. In some instances there are no other abnormalities, but often trigeminal paralysis, ptosis, strabismus, or weakness of the facial muscles has been recorded. A possible relationship between congenital alacrima and keratoconjunctivitis sicca was suggested by Sjögren,[3] who, upon reviewing the literature, found evidence of keratoconjunctivitis sicca in 5 of 14 cases. Since then Coverdale§ has reported four cases of congenital alacrima in which keratoconjunctivitis sicca subsequently developed. He suggests that in some of the adult cases of the latter disease a congenital defect in tearing is the underlying cause. He argues that the conjunctival and corneal changes become manifest in later life, when the tissues are no longer able to compensate for the drying that takes place.

In the cases of familial autonomic dysfunction which I have followed the conjunctiva has maintained a normal luster in spite of a more or less complete absence of tears. The stringy mucoid discharge so characteristic of keratoconjunctivitis sicca has been absent. The corneal changes, when present, are neither punctate nor filamentary but more closely resemble those seen in neuroparalytic keratitis.

Neuroparalytic Keratitis.—The initial corneal lesion in this disease, as well as in familial autonomic dysfunction, is a superficial, grayish infiltration which starts at the center and progresses peripherally. The epithelium soon begins to break down, and an indolent central ulcer is formed, which may go on to perforation. Associated with it is a relative absence of inflammatory signs, decreased lacrimation, and diminished corneal sensitivity. In neuroparalytic keratitis the alacrima and corneal hypesthesia are usually unilateral and there is a history of injury to the trigeminal nerve. These findings, along with an absence of the systemic manifestations of autonomic dysfunction, constitute the differentiating features.

Keratomalacia.—When corneal involvement occurs in infancy prior to the onset of systemic manifestations, familial autonomic dysfunction may be mistaken for keratomalacia (Case 3). In both conditions malnutrition is often pronounced, and a history of a difficult feeding problem is obtained. The corneal lesions in the two diseases may be similar indolent central ulcers. Alacrima is a constant finding in autonomic dysfunction and usually is present in xerophthalmia. However, the appearance of the bulbar conjunctiva is vastly different in the two conditions. In keratomalacia the initial changes take place in the conjunctiva. This membrane at first loses its luster, and then dry spots appear on it. This drying and wrinkling progresses until the entire surface of the globe assumes a greasy appearance. This condition is in direct contrast to the glistening, normal-looking conjunctiva present in autonomic dysfunction.

‡ References 3, 10, and 11.

§ References 10 and 11.

SUMMARY

Congenital alacrima and corneal hypesthesia are constant ocular findings in familial autonomic dysfunction—a disease characterized by drooling, blotching of the skin, intermittent hypertension, and emotional instability. The management of the indolent corneal ulcers, which occur in approximately one-third of these cases, is discussed, and three illustrative cases are reported.

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EYE MOVEMENT RESPONSES TO A HORIZONTALLY MOVING VISUAL STIMULUS

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EYE MOVEMENT responses to even the simplest visual stimulus moving in only one dimension in an otherwise structureless visual field are the result of activities at several levels. One may distinguish factors operating at three somewhat arbitrarily delineated levels: (*a*) factors involving the mechanical aspects of the orbit and the other levels intervening between the initiation of the nerve impulses designed to move the eye and the actual eye movement; (*b*) factors involving the operation of what are often called the psycho-optical reflexes, i. e., the mechanisms which make the eyes move so as to place and retain the image of a light stimulus constantly and accurately on the fovea; (*c*) factors operating at higher levels of complexity.

The first group of factors formed the subject of a recent study,[1] in which the conclusion was drawn that in an eye movement record of even reasonably high precision they introduce "errors" of generally negligible dimension and that, moreover, it seems possible to recognize as such the contributions to the eye movement record of these mechanical factors. When allowance has been made for these "errors," it follows that a comparison of the movements in the stimulus configuration and the concomitant eye movements will yield information concerning the mode of operation of the neural centers initiating eye movements.

There is some neurological evidence that the psycho-optical reflexes involve neural pathways different from those concerned with voluntary eye movements,[2] although the distinction between the two types of movement on this basis is by no means wholly clear.[3] It is possible, however, to postulate the existence of two levels corresponding to *b* and *c*, above, on entirely different grounds. The following experiment will illustrate this distinction.

Two light spots in a horizontal plane and separated by several degrees at the subject's eyes are alternately flashed on and off so that only one is exposed at any time; i. e., the light stimulus effectively jumps back and forth from one position to the other at regular intervals of about one second's duration. When the subject is first instructed always to fix the light which is currently exposed, he changes fixation about 150 msec. after the change in the light stimulus; i. e., the usual reaction time to a visual stimulus is exhibited. When the task has been continued for even as few as half-a-dozen of the rhythmic jumps, the subject changes fixation approximately synchronously with the change in the stimulus, and the eyes may actually reach the new position before the light has been flashed on there.

From the Ohio State University.

Based on research carried out in the Physiological Optics Laboratories, Ohio State University, which was supported in part by a grant-in-aid from funds allocated by the Ohio State University Research Foundation to the University in support of fundamental research.

Clearly the immediate visual stimulus is no longer initiating the response; a higher process, corresponding to level *c*, is playing the major role now.

The experiments to be described were carried out to elucidate the mode of operation of the second level, viz., the neural level concerned with the direct eye movement response to visual stimuli only and free as far as possible from the influence of such factors as memory and anticipation. It will become clear that the mode of operation of this level shows defined characteristics and that the distinction between the two postulated neurological levels also has a behavioral basis.

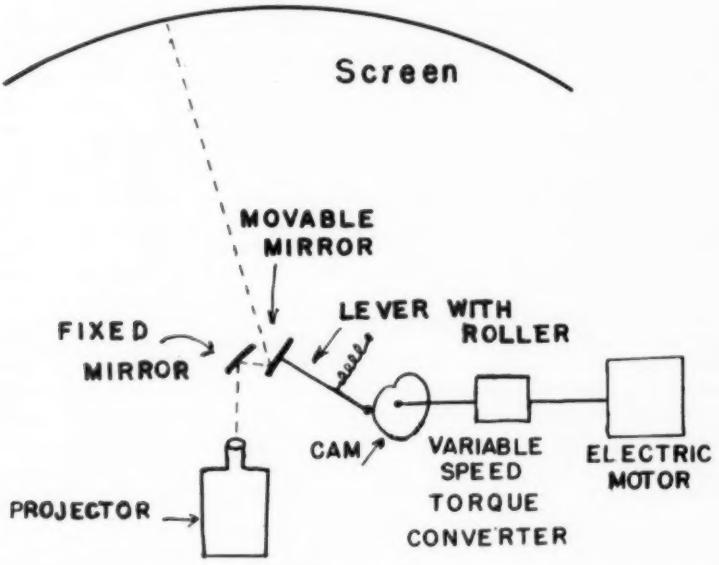

Fig. 1.—Diagram of apparatus for projecting a target moving with constant angular velocity.

It should perhaps be emphasized that this discussion of eye movements induced by moving visual stimuli does not make any reference to eye movements caused by some of the lower reflexes.

METHOD

Recording of Eye Movements.—A modified Ophthalmograph eye movement camera was used to register the traces of reflections of light sources in the corneas of the subject's eyes on continuously moving photographic film. By suitable devices, only the horizontal components of the eye movements were recorded, and the precision of the record remained within about 5 to 10 minutes of arc in the movement direction and about 1 msec. in the time dimension. By means of auxiliary light beams the position of the target was accurately registered on the film; in addition, a time scale was established on the record itself. Details of the method have been described previously.[1]

Stimulus Presentation.—For the presentation of step and pulse stimuli, two small neon light sources were placed in a completely dark room on an arc of 150 cm. radius centered on the subject's head. They were connected by the electric circuit described previously,[1] which allowed the extinction of one source synchronously with the lighting up of the other.

To generate visual stimuli moving with constant angular velocity across the visual field, the following apparatus was constructed (Fig. 1).

Light from a projector was reflected by a fixed mirror into a movable mirror and from there onto a large cylindrical screen of 150 cm. radius, at the center of curvature of which was situated the subject's head. This arrangement ensured that the stimulus light was at all times equidistant from the subject's eyes.

The movable mirror was mounted on a base which rotated around a vertical axis. A 12 in. (30 cm.) lever with a roller at its end was attached to the base, and the lever was held with a spring so that the roller moved along the edge of a cam, which was rotated at a constant speed for a given run. For different runs, rotation of the cam could be set at various speeds by adjusting the setting of a variable speed torque converter interposed between the cam shaft and a constant speed electric motor. The cam was constructed so that in one of its rotations the mirror was rotated in such a way as to cause the stimulus to move 30 degrees back and forth across the screen with constant velocity throughout each excursion.

As the mirror rotated, a drum mounted on the mirror shaft gave off or took up inextensible string which was wrapped around a rotatable metal cylinder and was held tight by having a weight attached to it. Rotation of the mirror was thus communicated with an extremely high degree of fidelity to the metal cylinder, on which was mounted a small thick piece of glass in the path of light rays emanating from a small streak-filament lamp operated on a 1.5 volt battery and brought to a focus in the plane of the film of the camera by a lens and a mirror. By proper choice of the size of the drum and cylinder, the thickness of the glass plate, and the axis of its rotation, it was possible to generate on the film a curve which precisely represented the movement that the eyes would have to make in following perfectly and synchronously the movement of the target across the screen. A time scale was established on the film either by interrupting the signal beam representing the stimulus every 1/100 second by a sector disc driven by a synchronous motor, or by recording the movement of one eye only and using the other eye tube to focus the image of a Strobotac on the film, flashing at the rate of 100 per second. A manually operated shutter allowed the light stimulus to the screen and the signal beam to the film to be exposed or intercepted simultaneously.

In order to record the eye movement response to other than constant velocity visual stimuli, a cam was constructed which produced continuous variations of the velocity of the stimulus moving across the screen. The variations were unpredictable to the subject and contained a number of reversals in direction.

It was possible to produce a horizontal sine motion of the target across the screen by the eccentric attachment on a rotating disc of an arm linked to the mirror turntable.

RESULTS

Eye movements records were obtained in response to the following types of visual stimuli:

(*a*) Instantaneous displacements of a light spot from one position to another in the same horizontal plane (step stimuli)

(*b*) Very brief displacements of a light spot from one position to another in the same horizontal plane with return to the original position (pulse stimuli)

(*c*) Light spot moving horizontally with constant angular velocities

(*d*) Light spot moving horizontally in an unpredictable fashion

(*e*) Light spot moving horizontally with the changes in velocity dictated by a trigonometrical sine function

(*f*) Light spot moving horizontally with constant angular velocity but exposed only intermittently

In all cases the light spot was the only stimulus in the subject's visual field which was completely dark, with the exception of the two lights in the far periphery

which were used to produce the corneal reflections. As far as possible the subject remained ignorant of the type of stimulus motion to be presented until the actual presentation took place. His head was firmly anchored by means of a bite board, and he was instructed to keep on looking at the light stimulus when it appeared.

Three young adults were used as subjects. None had a history of any ophthalmic or neurological disorder or showed any significant refractive error or muscular imbalance.

(*a*) *Step Stimuli.*—The response to the instantaneous displacement of a stimulus to another position in the visual field in the same horizontal plane is always a saccadic movement. The relation between the extent of the displacement and the magnitude of the movement is good, and if the original eye movement does not quite bring the image of the light spot back on the fovea, one or more corrective saccadic movements are usually executed.

There is always a reaction time of the order of 150 msec. between the displacement of the target and the onset of the movement response. The nature of saccadic movements has been explored in detail in a previous paper.[1]

(*b*) *Pulse Stimuli.*—Eye movement responses were studied to momentary displacements of a light spot for periods of 40 to 500 msec. The subject was instructed to keep his eyes on the light spot under all circumstances, and he was kept unaware of both the extent of the displacement and the exact moment at which it was to occur.

The response to a pulse stimulus always consists of (1) a saccadic movement, bringing the eyes fully over to a position corresponding to the displaced stimulus (the reaction time for this movement is the typical reaction time for a saccadic movement), and (2) a return sweep in the form of a saccadic movement, occurring either 200 to 250 msec. after the eyes have reached the new position or about 120 to 150 msec. after the stimulus has returned to its original position, whichever is the later. This is illustrated in Figure 2.

This response pattern may be interpreted as indicating that each displacement of the pulse stimulus is being reacted to individually. Thus, the initial displacement produces a saccadic movement after the usual reaction time, even though the stimulus may have returned to its original position before the first saccadic movement has begun.

(*c*) *Constant Velocity Stimuli.*—The subject's eye movements were recorded when the subject was instructed to look at the dark screen and suddenly a light spot appeared somewhere in the central region moving horizontally with a steady velocity. The spot was rectangular and subtended 10 minutes horizontally and 20 minutes vertically at the subject's eyes. Care was taken to randomize both the velocity and the position when the spot first appeared.

A typical record is shown in Figure 3. General conclusions concerning the response to this particular type of stimulus in three subjects follow.

There is a reaction time of 150 to 250 msec. before any response occurs. The reaction time seems to be longer when the velocity is slower. No attempt was made to study the relation between reaction time and difference between the positions of the eye and the spot when the latter first appeared.

When the velocity is below 25 to 30 degrees per second, there is usually a saccadic movement at the end of the reaction time which brings the eye onto the

target, and then the eyes start to move immediately with a velocity exactly that of the target. During the course of following the target there are often small saccadic movements of 1 degree, or sometimes more, back and forth around the target without any change in the velocity of the following movements. The latter are extremely smooth and constant in velocity in both eyes. When the velocity of the target is more than about 30 degrees per second, following movements are observed which are substantially slower than the target velocity and which are frequently interspersed with saccadic movements intended to reduce the constantly accumulating position error.*

During the course of following a constantly moving stimulus there are usually no changes in the velocity of the following movements. Even if the latter are too slow, the movements continue at the same speed but are frequently interrupted by saccadic movements to reduce the position error. Occasionally there are changes

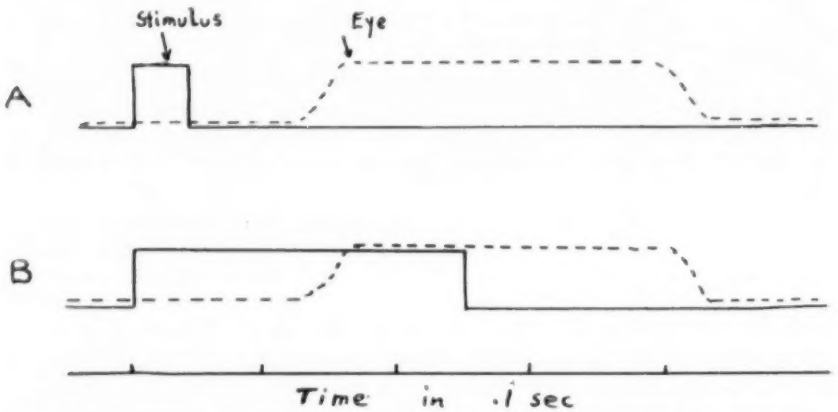

Fig. 2.—Response of the eyes to a momentary displacement of a stimulus light from one position to another one in the same horizontal plane (schematic). *A*, brief displacement (40 msec.); *B*, longer displacement.

in the velocity of the smooth movements, but they are not always in the direction of reducing the velocity error.

Similar results were obtained when the stimulus was stationary and then suddenly started to move with a constant velocity.

(*d*) *Unpredictable Movement.*—The presentation of unpredictable movements of the light spot on the screen was achieved by means of a special cam which produced a number of changes in direction, as well as many changes in velocity. The response to this type of stimulus presentation is characterized by the following features:

The tracking movements consist of constant-velocity movements interspersed with saccadic movements.

* The word error is used in this paper in the connotation it has in modern servomechanism theory, where it is used to describe the difference between the input and the output of a system.

Position error in the present context refers to a difference between the target position and the eye position, and velocity error means a difference between the velocity of the target and the velocity of the eye movement.

Changes in velocity of the smooth following movements are always discrete and occur at intervals of not less than 100 msec., and usually more. These changes are usually associated with small saccadic movements. Occasionally small sections of the records look as though smooth and rather slow changes in the velocity of the

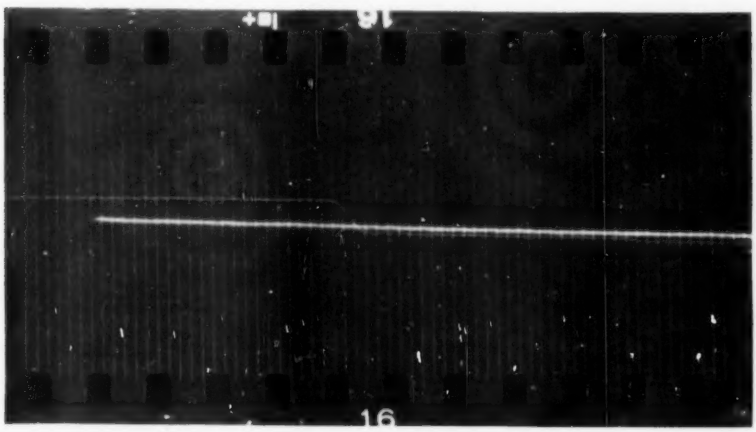

Fig. 3.—Photograph of eye movement in response to a target moving with a constant angular velocity of 5 degrees per second. Heavy trace, stimulus; light trace, eye movement; vertical lines, time, in 10 msec.

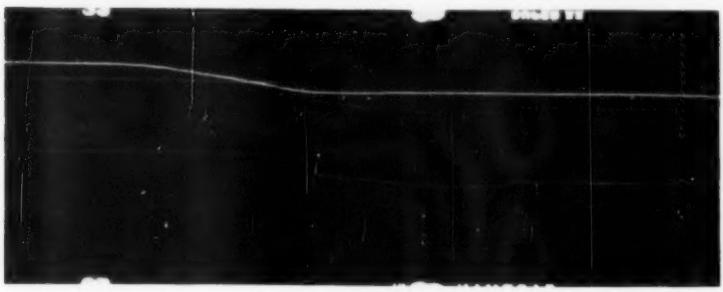

Fig. 4.—Binocular eye movement record during visual tracking of a light spot moving horizontally in an unpredictable manner. Length of record, one second. Separation of target and corneal reflection traces at beginning of record corresponds to accurate binocular fixation of target.

following movements had occurred. On careful analysis it is always possible to observe stretches of constant velocity movements separated by "knees," i. e., short periods of the order of 20 msec. during which a change had occurred from one velocity to another.

The lag between prominent changes in the stimulus velocity and the concomitant changes in the eye movement response is usually of the order of 150 msec.

Figure 4 is a typical example of eye movements during tracking of a visual stimulus moving in an unpredictable fashion. A single revolution of the cam lasted about 15 seconds. Subsequent revolutions repeated the movement pattern identically. It is of interest to note that the eye movement response to even the 20th presentation does not seem to differ significantly from that to the first presentation.

(*e*) *Sine Movement.*—On the first presentation of a horizontal sine movement of the target across the screen, the eye movements show all the characteristics enumerated under *d*, above; i. e., there are stretches of constant velocity following movements the velocity of which is changed discretely and which are interrupted by saccadic movements. However, records obtained after repeating a sine cycle about a dozen times show that a considerable amount of learning has taken place by then. There are then definite stretches of the record during which smooth and slow acceleration and deceleration of the eye movements are shown to occur. There is also little or no lag between the stimulus and the response, and occasionally the response even precedes the stimulus.

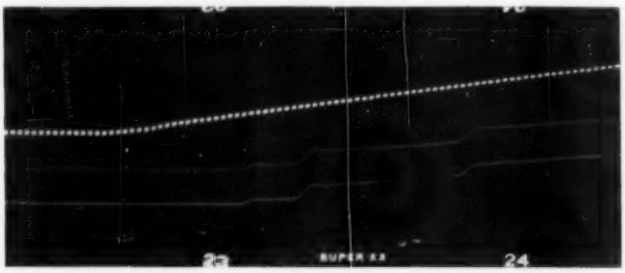

Fig. 5.—Eye movements induced by an intermittently exposed moving visual stimulus. Stimulus line corresponds to movement which the target would have made had it been continuously exposed. Actually it was exposed for 10 msec. every 120 msec. Separation of target and corneal reflection traces at beginning of record corresponds to accurate binocular fixation of target. Stimulus line is interrupted every 10 msec. Note smooth following movements after target had been moving for only about 300 msec., i. e., after, at most, three short flashes in spatial separation corresponding to movement had been presented.

It appears that for the amplitude of the sine motion used in these experiments, viz., 30 degrees, the maximum rate of presentation for which reasonably accurate tracking can be said to occur is about 3 cps.

(*f*) *Intermittently Exposed Moving Stimulus.*—The experiments described under *c* were repeated with an episcotister in place between the stimulus projector and the screen. In this manner it was possible to present a constant velocity stimulus exposed for only 10 msec. every 120 or 240 msec. At the usual target speeds the 10-msec. exposure is so brief that no movement of the target can be observed during the exposure time. One may describe the stimulus situation as that of 10-msec. flashes at 120- or 240-msec. intervals, each separated from the preceding one by the distance dictated by the speed of the movement.

Figure 5 illustrates that this stimulus situation gives rise to smooth following movements, just as though the target were actually moving continuously. However,

the correlation between target motion and eye motion velocity is somewhat poorer, and much more use is made of saccadic movements to reduce the position error than in situations in which the stimulus is continuously present.

COMMENT

It has become clear that the basic response patterns utilized in the operation of what have been called the psycho-optical reflexes are saccadic movements and constant velocity following movements. The basic nature of saccadic movements has been clearly understood since Dodge's conclusive investigations.[4] The exact mode of response to a moving stimulus, on the other hand, cannot be clearly ascertained from the available literature. Dodge appears to have believed that smooth changes in the velocity of eye movements (gradual accelerations) form a part of the basic response pattern, while others have suggested that even a constant velocity movement is not encountered in an original, unlearned response.

The experiments described in this paper allow the following general conclusions to be drawn concerning the eye movement response to a simple moving visual stimulus under conditions in which there is no reason to expect the involvement either of a lower reflex arc or of any anticipatory process.

Fig. 6.—Eye movements in response to a brief (200 msec.) constant velocity stimulus.

Movement of the target across the retina or the illusion of movement as produced by short bursts of retinal stimulation in the temporal and spatial sequence of a typical movement stimulus, but separated by reasonably short time intervals, induces a regular constant velocity movement in both eyes. The velocity of this movement is generally, but by no means always, closely related to the speed of the target, and within a reasonable span of target velocities the eyes start moving with the correct velocity after a reaction time of often less than 200 msec. After the stimulus has ceased to move or has disappeared from view, the eyes continue to move for 100 msec. or more.

This effect is well illustrated in Figure 6, which shows the response to a brief constant velocity stimulus which had already disappeared before the eyes started responding. There is a saccadic movement to a position which the target would have occupied had it not been withdrawn from view. The smooth following movement induced is of the correct velocity and persists for nearly 200 msec. before it is checked. Several hunting saccadic movements follow.

During tracking, adjustments to errors are carried out by discrete changes in the velocity of the following movements and by saccadic movements. A great deal of the adjustment of errors due to an incorrect velocity of the constant velocity following movements, as well as the adjustment of all position errors, takes place by means of saccadic movements.

Changes in the velocity of the eye movements during visual tracking always takes place discretely. Smooth changes in velocity, i. e., gradual accelerations or decelerations, are found only in situations in which there is reason to believe the processes of anticipation or prediction to have been in operation.

One may picture the mechanism of eye movement responses to moving visual stimuli as a perception of movement, the putting into operation of a well-integrated train of impulses designed to impart to the eyes a constant velocity movement, the correction of position errors by saccadic movements, and the modification of the speed of the following movements in discrete steps as the need arises. The responses all lag behind the stimulus by an interval of the order of a reaction time.

This is undoubtedly a closed loop or feedback circuit in which errors are used to modify the performance, but it is a feedback system of a higher order that uses both jump responses and smooth constant velocity responses, all of which are used and modified discretely and at intervals. To elucidate further the nature of this feedback system, it would be necessary to investigate the magnitude of the errors which induce certain responses and, in a more general way, to correlate the stimulus with the eye movement response.

An interesting confirmation of one of the findings in the present experiments, viz., that the maximum velocity of a moving stimulus that is accurately followed by the eyes is about 30 degrees per second, is contained in a recent report by Ludvigh and Miller.[5] These authors measured the visual acuity of a number of subjects for targets moving with constant angular velocities ranging from 10 to 170 degrees per second. They found that no significant decrement occurs in the visual acuity for target velocities of up to 20 to 50 degrees per second (in some cases even to 80 degrees per second), but that for higher target velocities a progressive diminution in the visual acuity becomes evident in all cases. It seems clear that so long as the target velocity can be matched with reasonable accuracy with a constant velocity eye movement, a steady retinal image will prevail, permitting good resolution. Once the target velocity is increased beyond this range, however, one would expect a progressive reduction in the visual acuity.

SUMMARY

A number of eye movement responses to moving visual stimuli have been recorded photographically under conditions in which it is reasonable to expect the exclusion of factors involving the higher perceptual levels, as well as of the lower reflex arcs. The inference is drawn that these responses give information concerning the mode of operation of what are often called the psycho-optical reflexes.

It appears that two basic response patterns are utilized: saccadic movements and contant velocity following movements. The latter are initiated by a movement in the visual field or the perception of movement. In the original response to a moving stimulus both types of eye movements appear, and they are changed discretely and at intervals as differences between the target direction and the eye position develop. When the target position is changed instantaneously from one position to another, the response consists only of saccadic movements. All responses lag behind the stimulus by an interval of the order of a reaction time. As the subject becomes familiar with the target motion, he is able to anticipate the latter. The

reaction time then becomes eliminated, and gradual accelerations of the eye movements become apparent which do not otherwise seem to form a part of the basic response pattern.

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RELATIONSHIP OF HYPERCEMENTOSIS TO THE EXOPHTHALMOS OF HYPERTHYROIDISM

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HYPERCEMENTOSIS is a dental condition characterized by an "abnormal thickening of the cementum. It may be diffuse or circumscribed, i. e., it may affect all the teeth or only certain parts of one tooth." [1] The root of a tooth (Fig. 1) consists of a root canal (*A*) surrounded by dentine (*B*). Immediately around the root, and entirely surrounding it, is a thin even black line, the peridental membrane (*C*). Immediately surrounding the peridental membrane is a thin even white line, the lamina dura (*D*), which represents the dense plate of bone lining the socket of the tooth. Just beneath the peridental membrane is the thin layer of cementum. Hypercementosis (Fig. 2) is diagnosed roentgenographically by the increased deposition of cementum immediately beneath the peridental membrane (*A*) and the bulging of the peridental membrane and lifting of the lamina dura from the surface of the root at the point of increased cementum deposit (*B*). The etiology of hypercementosis has been explained by a hereditary factor,[2] abnormal stress,* a defense reaction of the organism to a low-grade irritation in pulpless teeth,† and an inherent tendency in certain persons, rather than a hyperplastic response to infection only.[4] Some cases are the result of local pathology (Fig. 3). However, in a number of cases of hypercementosis no explanation as to the etiology is apparent.

PRESENT INVESTIGATION

In a previous communication,[5] it was pointed out that hypercementosis was found in 20% of a group of normal white women, while occurring in about 80% of a group of thyrotoxic women of the same age distribution. The present investigation was undertaken to determine whether this easily recognized dental condition is related to the occurrence of malignant exophthalmos which complicates some cases of hyperthyroidism. To this end, a group of normal controls; a second group, of thyrotoxic patients without malignant exophthalmos, and a third group, of hyperthyroid patients with malignant or progressive exophthalmos, were examined for the presence of hypercementosis. The material for this study and the results follow.

1. *Controls.*—A. Forty-two consecutive full-mouth dental x-rays were obtained from the dental clinic of The Johns Hopkins Hospital. In 12, or 32%, of cases there was evidence of hypercementosis. Two cases could be explained by local

From the Wilmer Ophthalmological Institute of The Johns Hopkins Hospital and University.

* Gottlieb, B., cited by Boyle, P. E., in Kronfeld.[3]

† Boyle, P. E., in Kronfeld,[3] p. 267.

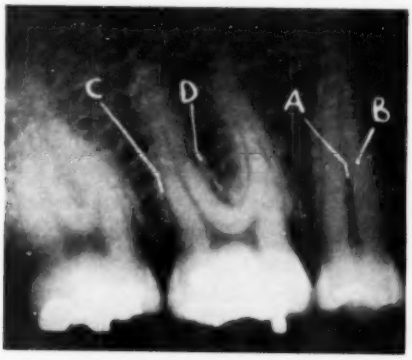

Fig. 1.—Normal root.

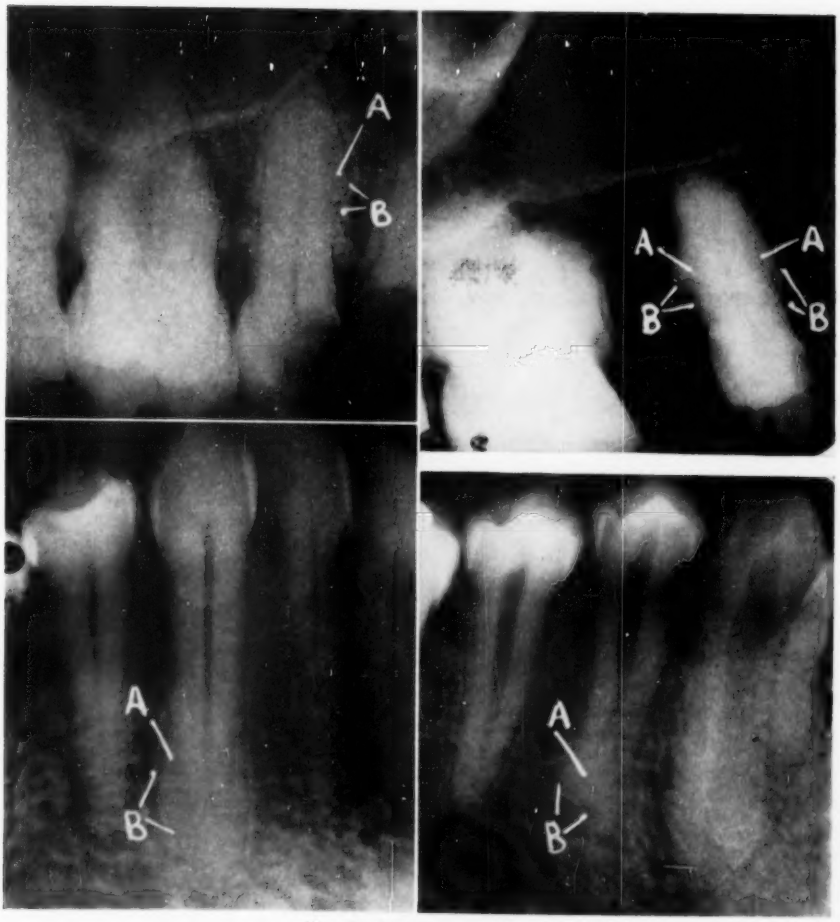

Fig. 2.—Hypercementosis.

pathology. An analysis of the other 10 cases in which no local cause for the hypercementosis could be found revealed previous operation for toxic diffuse goiter in 4 and for nodular goiter in 1. These five cases were therefore excluded.

B. Fifty patients with no known history of hyperthyroidism were selected at random from the outpatient department of The Johns Hopkins Hospital. Full-mouth dental x-rays of these patients showed 15 instances of hypercementosis, 6 of which could be explained on the basis of local pathology. An analysis of the information available from the histories in the other nine cases showed no evidence of hyperthyroidism in seven and an impression or suspicion of hyperthyroidism in two. These last two cases were excluded.

Summary of Control Groups: In 92 unselected control cases there was an over-all incidence of hypercementosis of about 30%. An analysis of the 27 proved cases showed either definite or suspected hyperthyroidism in 7. When these cases were removed from the series, there remained 20 instances of hypercementosis in 85 non-

Fig. 3.—Hypercementosis with local pathology.

hyperthyroid persons. Eight, or 10%, could be explained on the basis of local pathology, while in the remaining 12, or 14% of the whole, there was no explanation for the hypercementosis.

2. *Hyperthyroid Patients Without Malignant Exophthalmos.*—This group consisted of 49 patients with known hyperthyroidism having approximately the same age, sex, and color distribution as the control Group B. None of the patients in this group had progressive or malignant exophthalmos. In this group, there were 32 cases of hypercementosis, an over-all incidence of 65%. Three cases (6%) could be attributed to local pathology. In the remaining 29 cases there was no factor other than the hyperthyroidism to which the hypercementosis could be attributed. This incidence (60%) contrasts with an incidence of 14% in the nonhyperthyroid control group.

3. *Hyperthyroid Patients with Malignant Exophthalmos.*—This group consisted of 15 patients, in 11 of whom the progressive exophthalmos developed after thyroidectomy—the so-called malignant exophthalmos group. In this group, there

were three cases of hypercementosis, an over-all incidence of 20%. Two cases, or 14%, could be explained on the basis of local pathology, while in one there was no obvious explanation.

These foregoing results are shown in the accompaning Table.

Incidence of Hypercementosis in Relation to Hyperthyroidism With and Without Malignant Exophthalmos

Group	Cases Total	Incidence of Hypercementosis		Incidence Attributable to Local Pathology		Incidence of Unexplained Hypercementosis	
		No.	%	No.	%	No.	%
Nonhyperthyroid controls	85	20	24	8	10	12	14
Hyperthyroid without malignant exophthalmos	49	32	66	3	6	29	60
Hyperthyroid with malignant exophthalmos	15	3	20	2	14	1	7

According to the data above, hypercementosis not explainable on the basis of local pathology occurs about four times as frequently among hyperthyroid persons without the progressive type of exophthalmos as it does in either the control groups or in patients with progressive or malignant exophthalmos, the incidence being statistically the same in the latter two groups. It would seem that the absence of hypercementosis in an hyperthyroid patient might be regarded as evidence for the possibility of development of malignant exophthalmos, especially in the face of other indicative symptoms.

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Annual Reviews

GLAUCOMA

Review of the Literature for 1953-1954

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II. Congenital Glaucoma
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THE YEAR 1953-1954 has provided many original and exciting contributions to our knowledge of glaucoma. As in the past, the table of contents indicates the sequence in which these contributions will be considered.

I. THE PRIMARY GLAUCOMAS

A. Classification

The previous glaucoma review emphasized the opinion that the primary glaucomas are composed of at least two distinct forms, i. e., narrow-angle (iris-block or congestive) glaucoma, and the wide-angle (open-angle, non-iris-block, noncongestive, or chronic simple) glaucoma. Assuming that further distinctions may be made as our knowledge increases, the originator of this classification, Otto Barkan,[1] presents an exceptionally complete and comprehensive discussion of the evidence to

support the classification. The article discusses not only the time-honored distinctions, such as symptomatology, anterior chamber depth, corneal diameter, refraction, and response to therapy, but also the confirmative evidence supplied by more recent techniques, such as tonography. Barkan summarizes his distinction by defining narrow-angle glaucoma as a glaucoma caused by closure of the angle by the root of the iris. This glaucoma is the result of increased functional seclusion of the pupil, iris bombé, narrowing of the angle, and, finally, angle closure, associated with secondary expansion of the vitreous chamber and increased anterior displacement of the lens.

In wide-angle glaucoma it is his feeling that the impediment to outflow is in or near the angle wall (trabeculum-Schlemm's canal mechanism). The angle remains open, and the axial depth of the anterior chamber does not change during the course of the disease.

Although not all writers would agree with Barkan's suggested pathogenesis, there is other evidence to support the division of the primary glaucomas into two types. Morpurgo [2] studied 23 cases of chronic glaucoma and 6 of the acute type and concluded that there was a considerable difference between chronic simple glaucoma and acute glaucoma. He is of the opinion that acute glaucoma is principally functional in nature and consists of a vascular instability, resulting in acute arterial and capillary vasodilation, which causes edema of the ciliary body and blockage of the chamber angle. Chronic glaucoma is probably the result of a complex of alterations in the anatomy of the eyeball.

Further evidence is advanced by Kleiner,[3] who studied the aqueous veins with an infrared light after the injection of fluorescein into the anterior chamber. He found that during an acute attack of narrow-angle glaucoma the aqueous veins were reduced in number and their caliber was reduced, indicating a block to the outflow of aqueous. When the tension was restored to normal, the aqueous veins returned to normal, suggesting a removal of the block. However, in wide-angle glaucoma there was no alteration in the aqueous veins under pressure.

Duke-Elder [4] agrees that there is a congestive form and a noncongestive form of glaucoma but reiterates his belief that the early stages of glaucoma of both types are characterized by a condition of vascular instability, periodic phases of sympatheticotonia, wherein a raised venous capillary pressure occurs, followed by phases wherein circulatory control reasserts itself. He suggests that the primary fault in congestive glaucoma is an intense vasodilation of the whole uveal circulation, associated with an enormously increased capillary permeability and swelling of the tissues, but that in chronic glaucoma the variation in intraocular pressure must be in the drainage channels and follows chronic venous stasis.

B. Mechanism

1. *Narrow-Angle Glaucoma.*—The concept that narrow-angle glaucoma is a local ocular disease has received considerable written support. Barkan [5] amplifies his beliefs that pupillary block (functional seclusion of the pupil, or physiological iris bombé) is probably the main factor in the local pathogenesis of narrow-angle glaucoma.

He states that with age or certain abnormalities the axial depth of the anterior chamber decreases, bringing more of the iris in contact with the lens and thereby increasing the pupillary block (the term applied to the resistance to the flow of aqueous from the posterior to the anterior chamber produced by the iris). This block will, in turn, increase the pressure in the posterior chamber, resulting in an increased pressure in the vitreous compartment. The vitreous pressure would then be greater than the pressure in the anterior chamber and would push the lens zonule diaphragm forward. Thus, the two factors will be self-intensifying, inasmuch as the narrowing of the anterior chamber will cause increased pupillary block, which will cause further narrowing of the anterior chamber, resulting in a vicious circle.

It is suggested that another factor may be that the pupillary block and the iris bombé, by exerting a forward and outward pull, rotate the ciliary body on its axis. As a result, the insertion of the zonule advances, permitting an anterior displacement of the lens. This would also increase pupillary block.

Further information regarding the pathogenesis of the anatomically narrow angle comes from Törnquist,[6] who attempted to establish the importance of genetic factors in determining the magnitude of the chamber depth and felt justified in advancing the hypothesis that the shallow anterior chamber of acute glaucoma is genetically controlled. Consequently, he assumed that the shallow chamber is a developmental peculiarity which is a preexisting and predisposing factor in acute glaucoma. In his studies he used an improvement of Lindsted's instrument to measure the depth of the anterior chamber described by Stenström.[7]

That the size of the pupil plays an important part in the closure of the narrow angle is made clear by the gonioscopic studies of Redmond Smith,[8] who described the opening and closing of the angle of the anterior chamber by the iris root in perfect harmony with the pupillary constriction and dilation caused by the gonioscopic light. He feels, quite properly, that this phenomenon might be used to great advantage in the diagnosis of narrow-angle glaucoma.

Further evidence that pupillary dilation may precipitate an attack of narrow-angle glaucoma is demonstrated by the report of Morate,[9] who tells of a case of acute monocular glaucoma in a woman 26 years old precipitated by the internal consumption of atropine for the relief of asthma. In another report, Barkan [10] reports an unusual patient with spastic miosis in the left eye. The patient developed an uncontrollable tension which required iridectomy in the right eye, but the eye with the spastic miosis has been followed for eight years without an attack.

That narrow-angle glaucoma is a purely local disease is objected to by Weinstein,[11] who cites many cases of acute glaucoma in which the attack is simultaneous with an acute emotional shock. Whether these sympathetic shocks precipitate the attack through their effect on the pupil is not determined, but a most interesting case is presented.

O.B., male, 40 years old. Chronic congestive glaucoma. Occasionally severe depression. The ocular tension is not influenced by pilocarpine, but well influenced by physostigmine (eserine). The patient always asks for physostigmine instead of pilocarpine and watches the flask used. Once we exchanged the labels of the two flasks, so that the patient actually received physostigmine but supposed it to be pilocarpine. Shortly afterwards, tension increased.

Weinstein also stresses the tension-lowering effect of frontal lobectomy in normal subjects to illustrate the control of the central nervous system over intraocular pressure.

2. *Wide-Angle Glaucoma.*—That wide-angle glaucoma still remains a disease of unknown etiology is suggested by the following paragraphs. The diversity of ideas presented can only emphasize the lack of conclusive evidence. For both the believers in the mechanical theory of glaucoma and the disbelievers, this reviewer would like to recommend the text of Weinstein [12] on "Glaucoma." This book is a presentation of the diagnosis, etiology, treatment, and pathology of glaucoma which includes reference to the mechanical school but primarily reflects the views of the author, who is one of the foremost exponents of the neurovascular school. It is a thought-provoking volume, containing so much information that it is quite impossible to review it in this article.

(*a*) Hypersecretion: This factor has not been advocated as a cause of primary glaucoma in the literature of 1953-1954.

(*b*) Aqueous Retention: It is apparent that the majority of investigators believe wide-angle glaucoma to be due to an impediment to the outflow mechanism. If one traces the routes by which the aqueous leaves the eye to enter the general circulation, one is impressed by the complexity of the mechanism, and one can easily see why so many beliefs are prevalent.

Not knowing which belief or beliefs are true, the reviewer will subdivide the material into the area or system that the author tends to incriminate as the site of outflow block.

(1) Trabeculum. Grant and Trotter [13] state that in primary open-angle glaucoma, in which no abnormality is visible in the angle, it seems that the impediment to outflow must be explained by abnormality of the aqueous-outflow channels themselves or by elevated venous back-pressure in the recipient vessels. The evidence is principally in favor of an abnormality of the outflow channels. The same kind of tonographic finding of resistance is obtained in primary open-angle glaucoma as in the glaucomas due to obvious mechanical obstruction. In some of these other glaucomas the resistance is found to persist after enucleation, with the venous back-pressure zero.

(2) Aqueous veins. Ashton,[14] continuing his studies of Schlemm's canal and the aqueous veins, demonstrates his findings of the arterial relations to Schlemm's canal. By actual injection technique of the canal of Schlemm and the ophthalmic artery, he was unable to prove any actual arterial connections with the canal, although an incomplete arterial circle was demonstrated in close proximity to it. He also showed that the aqueous channels were not found in company with the blood veins, but that they ran alone or in company with arteries.

Stepanik,* by a method of artificially produced projectiles of blood, estimated the rate and quantity of visible outflow in the aqueous veins and found it less than the total outflow. It was assumed that the rest of the aqueous was concealed by blood and therefore could not be measured.

Hodgson and MacDonald,[17] attempting to correlate the flow of aqueous in the aqueous veins with the intraocular pressure, used the fluorescein instillation test. In glaucomatous eyes they noted that the fluctuations in the bulk rate of flow, either spontaneous or after the instillation of miotics, were reflected in changes in flow of clear fluid in the aqueous veins. However, neither the fluctuations nor the changes in the aqueous veins had a constant parallel relation to the tonometric readings.

* References 15 and 16.

(3) Venous pressure. Bain,[18] attempting to determine the relationship between the episcleral venous pressure and the intraocular pressure, found that changes in the episcleral venous pressure generally seemed to precede changes in the intraocular pressure and that these changes occurred bilaterally. Using this information, Bain suggested that changes in the venous pressure might act as the trigger mechanism to start a pathological rise in pressure in narrow-angle glaucoma.

Confirming the effect of episcleral venous pressure on the intraocular pressure, Huggert,[19] by placing contact lenses on normal and glaucomatous eyes, produced a not insignificant rise in the intraocular pressure. It was of interest that the rise was higher in older patients and that in them it took longer for the pressure to return to normal.

Weinstein and Forgács [20] believe that changes in venous pressure in the body, as well as in the episcleral veins, are controlled by the central nervous system. They were able to correlate the pressure of the cubital vein with the filling of the episcleral-aqueous veins. In contrast to the normal, the venous pressure in the glaucomatous subject was higher in the morning, an observation which would explain the usual glaucoma diurnal curve.

(4) Hypothalamic region. Thiel [21] and Magitot [22] both emphasize the control of the intraocular pressure by the vegetative nervous system of the body. Both feel that the disturbed ocular tension must be considered the result of local, endocrine, and nervous factors.

(5) Sympathetic system. Cristini and Strazzi [23] decreased the daily tension variations in chronic glaucoma by stellate ganglion block and felt that this diminution was due to a lack of sympathetic stimulus to the small uveal vessels. Thus they postulate that the increased tensional variations in glaucoma are due to an alteration in the cervical sympathetic system.

(6) Allergy. Because the histamine content of the blood in glaucomatous subjects is increased in chronic glaucoma before operation and is normal after, Fontana [24] thinks that allergic disease may have some importance to glaucoma.

(7) Endocrine system. The possibility of endocrine control of intraocular pressure is considered by Radnót and Orbán,[25] who found homolateral elevation in intraocular pressure in two of seven rabbits following ligation of a deferent duct.

(8) Miscellaneous factors. Mather and associates [26] and Nakaji [27] found considerable alteration in intraocular pressure following electroshock treatment. Nakaji found only an increase in tension, but Mather found a fall unless the patients were pretreated with curare. Then there was a marked rise, which rapidly fell to normal. Interestingly, Mather postulates that the alteration is not directly through neurological mechanisms but is due to the effect electroshock has on the cellular and extracellular fluid osmolarity, which in the eye may affect the osmotic gradient to attract fluid from the globe.

Manning and Hollander [28] suggest that care be taken in the administration of electroshock to glaucomatous patients by citing flare-ups in two eyes.

3. *Low-Tension Glaucoma.*—Low-tension glaucoma exists and is due to constriction of the nutrient vessels of the optic nerve, according to Vail [29] and Palich-Szanto.[30] Palich-Szanto emphasizes the rapid deterioration of the fields due to the

involvement of these posterior vessels. Becker and Friedenwald [31] found that the aqueous outflow when measured by tonography is reduced in low-tension glaucoma, suggesting anterior involvement as well.

4. *Malignant Glaucoma.*—This excruciating situation is dramatically described by McDonald,[32] who recommends lens extraction for its control. Shaffer [33] feels that the pathogenesis of malignant glaucoma may be similar to the condition in some aphakic eyes or eyes with subluxated lenses in which synechias form between the iris and the vitreous face, producing a pupillary block. Aqueous collects in the posterior chamber, detaching the vitreous anteriorly, or passes behind a posteriorly detached vitreous body, shoving it forward, producing glaucoma. The anterior chamber can be either collapsed or filled with vitreous. Iridectomy will usually result in a cure of this type of glaucoma.

A similar syndrome is sometimes seen after lens extraction is done to relieve a malignant glaucoma. It becomes impossible to refill the anterior chamber because aqueous runs posteriorly, forcing detached vitreous and iris forward and continuing the malignant course. Only by breaking the pupillary block can this type of glaucoma be relieved. If the aqueous is collecting anteriorly, iridectomy will be helpful. If aqueous is collecting behind the detached vitreous, the only chance of cure is actual incision through the detached vitreous to break the posterior anterior pupillary block.

C. Diagnosis

1. *General Considerations.*—The obligation of the ophthalmologist in glaucoma diagnosis can never be overemphasized. The latest reminder of our responsibility comes from Philadelphia, where, in a screening test of 9,645 persons, Hankla [34] states that 2.2% had a glaucoma diagnosis. Sugar [35] discusses the early diagnosis of glaucoma, emphasizing the provocative and biomicroscopic signs. He calls attention to the possibility of the superimposition of acute narrow-angle glaucoma in a preexisting wide-angle type.

In a general discussion of the differential diagnosis of narrow- and wide-angle glaucoma, Weekers and Prijot [36] emphasize the importance of gonioscopy, tonography, and tension curves. Of interest is the report of temporary color blindness as a result of acute glaucoma,[37] as well as Sarkies'[38] survey, in which, contrary to general belief, he did not find glaucoma more frequent in the Gold Coast African.

2. *Tonometry.*—The publication of the report of the Committee on Standardization of Tonometers by Friedenwald and associates [39] can evoke only admiration for the careful and thorough work that it contains. One finds contained in this publication the fundamental work for the calibration of tonometers, the basic calculations upon which flow and outflow of aqueous are determined, and the basic work on scleral rigidity. Of new interest to most will be the nomograms for determining both the scleral rigidity and the intraocular pressure of eyes with varying sizes and curvatures of the cornea. It is anticipated by this reviewer that this work will become the standard reference for tonometry for years to come.

The splendid work accomplished by this Committee has provided the inspiration for a similar one in Great Britain.[40]

3. *Provocative Tests.*—(*a*) The Dark Room Test: In 117 normal eyes, Higgitt [43] found that one hour in a darkroom produced a mean rise of 2.1 mm. Hg, the high being 9 mm. (Schiøtz). In chronic simple glaucoma the rise was similar, with

a mean of 2.1 mm. Hg and a high of 14 mm. Hg (Schiøtz). However, in 122 eyes with congestive types of glaucoma the mean rise was 16.2 mm., with a high of 64 mm. Hg. He concluded that the test was of value only in congestive glaucoma and that 75% of these cases gave a positive result. Interestingly, he reported two cases with a positive darkroom test that were negative to mydriasis produced by epinephrine. To explain the rise in tension, Higgitt hypothesized a neurovascular mechanism to produce the small rise in normal eyes and in eyes with wide-angle glaucoma but felt the large rise in congestive glaucoma was due to angle closure.

(*b*) The Water-Drinking Test: Leydhecker,[44] who has written extensively on the provocative tests, found that of 264 normal eyes, 12 had a rise of 6 mm. Hg, 2 a rise of 7 mm. Hg, 1 a rise of 8 mm. and 1 a rise of 10 mm. He states therefore that only rises of 10 mm. Hg (Schiøtz) or more can be regarded as pathological. He further states that of 168 glaucomatous eyes with an initial pressure of 30 mm. or less the incidence of positive tests was between 17% and 25%. If 8 or 9 mm. is taken as a positive response, then only 32% of those with an initial pressure below 30 mm. were positive. He thus concludes that the test is unreliable.

(*c*) Vasculat. Leydhecker [45] describes a new provocative test which he claims has a reliability of almost 90%. This test makes use of a new vasodilator, Vasculat, which is injected subconjunctivally, allowing the eye to be tested directly, with the fellow eye serving as a control. A positive result is one in which the rise of tension is more than 12 mm. He feels the action is the result of an acute and intense dilation of the episcleral and conjunctival vessels, causing the aqueous veins to fill with blood. This reflux of blood will throw an eye with poor facilities for outflow out of balance.

(*d*) Anterior Chamber Puncture Test: In discussing Verrey's lack of confirmation of his data, Kronfeld [46] reestablishes his findings that a significant difference in response to the anterior chamber puncture was found to exist between eyes affected with early chronic open-angle glaucoma and nonglaucomatous (control) eyes. He, however, wholeheartedly agrees with Sugar's statement that "the anterior chamber puncture test is an excellent test which is useful in investigation but rarely indicated in office practice."

(*e*) Compensation-Maximum Test: Kleinert observed a retrograde influx of blood from episcleral veins into the aqueous veins during compression of the eyeball in different types of glaucoma. The dynamometer pressure necessary to produce this phenomenon was termed compensation maximum. In normal eyes compression of the eyeball up to 150 gm. usually would not produce this backflow, but in glaucomatous eyes in many instances a pressure of 100 gm. or less sufficed to force blood back into the aqueous veins. In the opinion of Stambaugh and associates,[47] the compensation-maximum test most frequently yielded high values in normotensive eyes and low values in glaucomatous eyes, but they did not feel it sufficiently correlated or characteristic to be of value in the clinical differentiation of these two groups.

(*f*) Amyl Nitrite Test in Primary Glaucoma: Cristini and Pagliarani † postulate the vascular nature of glaucoma. They noted the effect on ocular tension of the amyl nitrite test on 50 eyes in basal conditions, after the instillation of cholinergic drugs, and after unilateral alcohol-procaine block of the stellate ganglion. In general, a

† References 48 and 49.

fall of ocular tension was observed, more markedly in early cases and when the initial tension was high. In the more advanced cases and after the instillation of cholinergic drugs, the fall was low, or even lacking. Cristini thought that the fall in basic intraocular pressure was caused by a diminution of the hydrostatic capillary venous pressures in the uvea following widening of the capillary network. The rate of decrease, moreover, may be related to the widening capacity of the uveal capillary network, and thus it follows that the response to the amyl nitrite test may provide useful evidence for the clinical evaluation of the severity of this fundamental symptom in primary glaucoma.

4. *Tonography.*—Becker and Friedenwald [31] summarize the methods of estimating aqueous outflow. They feel that Goldmann's fluorescein method has the advantage of an undisturbed eye but the disadvantage of requiring expensive and elaborate apparatus. Grant's method of tonography contains some possible sources of error, such as whether the aqueous actually has a steady state of flow, whether the blood volume is constant, whether the episcleral venous pressure is the same during tonography as in the undisturbed eye, whether the facility of outflow in the eye at higher pressure from tonography is the same as in the undisturbed eye, and whether variations from average values for corneal curvature and scleral rigidity affect tonographic measurements. They provide some of the answers by discussing the Traube-Hering waves (slow, wave-like variations in blood pressure which are reflected in the intraocular pressure) and the fact that the application of the tonometer sometimes causes a steep diastolic rise in blood pressure, and they state that when scleral rigidity is high it can produce a 10% variation in coefficient of outflow. The effects on tonography of the provocative tests, miotics, surgery, the menstrual cycle, and pregnancy are discussed.

In separate papers, Horwich and Breinin [50] and de Roetth [51] discuss the relationship of tonography to the phasic variations of the intraocular pressure. The former authors are of the impression that the rising phase of ocular tension is usually accompanied by a decrease in the facility of aqueous outflow, but de Roetth concluded that the facility of outflow did not change considerably, while at the same time the intraocular pressure varied markedly. That some diversity is to be expected in studies on tonography can be readily seen when the possible sources of error, as advanced by Becker and Friedenwald, are studied.

Mansheim [52] discusses the coefficient of outflow in such unusual types of secondary glaucoma as the Sturge-Weber syndrome, buphthalmos, etc.

Finally, for those who are interested in a simple introduction to tonography, the pamphlet written by Ballantine [53] has been reprinted.

Blaxter [54] feels that the bulbar pressure test has several decided advantages over tonography, the principal one being that it gives a higher percentage of positive results in early cases of glaucoma when the test is used for diagnosis. This higher percentage is predicated on the assumption that it requires about 45 gm. of pressure to force aqueous through all the possible exit channels, and that therefore tonography is done without the trabeculum being put under stress. The bulbar pressure test is performed by applying a Bailliart dynamometer to the eye behind the insertion of the lateral rectus muscle and applying a force of 50 gm. After allowing 10 to 15 seconds for the intraocular pressure to stabilize, the reading *B* is noted. This position is maintained for four minutes, after which the reading *C* is taken. The dyna-

mometer is then removed to give the reading *D*. The fall in pressure is represented as a percentage of the initial tension, i. e.,

$$\frac{B\text{–}C\ (100)}{B} = \text{the outflow fraction (O.F.)}$$

Blaxter states that on normal eyes the average outflow fraction was 34 mm. Hg and that therefore values below 29 mm. are abnormal, whereas values below 25 mm. are definitely abnormal. When the pressure was below 29 mm. Hg, 88% of the outflow fractions were positive, and when the pressure was below 25 mm. Hg, 73% were positive.

5. *Perimetry.*—Magitot [22] discusses his belief that glaucomatous optic atrophy and the tension are independent of each other and are governed by neurovascular maladjustment. He cites the presence of arterial and capillary obliteration in the retrobulbar parts of the optic nerve, which he feels could not be caused by the raised intraocular pressure.

The fact that Goldmann's artificial scotoma could be obtained by the commonest provocative tests is discussed by Ferraris de Gaspare,[55] who found the enlargement of the scotoma to be directly proportional to the tonometric increase produced by the test. The writer felt that the variations in the scotoma were much more significant than the angioscotoma and that it may constitute a useful criterion for an early diagnosis of chronic glaucoma.

The parallelism beween the optic nerve changes and the visual field changes in glaucoma are pointed out by Funder.[56] Day and Scheie [57] call attention to the simulated field changes in glaucomatous patients caused by ciliary spasm, miotics, and cataract.

A small scotoma detector, resembling a miniature mechanized tangent screen, which can be used by the general physician is described by Williamson-Noble.[58]

6. *Phasic Variations.*—The diagnostic virtues of the daily tension variations are elucidated by Matteucci,[59] who found that the normal variation is about 10 mm. (Schiøtz) during the day. In glaucoma the base curve is elevated.

7. *Gonioscopy.*—The virtues of gonioscopy in subjects suspected of having tumors of the posterior chamber are extolled by Scheie.[60] With the pupil well dilated, the writer found the commonest space-taking lesion of the posterior chamber to be the intraepithelial cyst of the iris or ciliary body. These lesions have a characteristic appearance that differentiates them from malignant tumors, which show a more granular surface and a strong tendency to invade the angle of the anterior chamber and the root of the iris. Moskowitz [61] reports two cases of undiagnosed uveitis which were found to have foreign bodies by the use of gonioscopy.

A four-sided modification of this Gonioprism is reported by Lee Allen and associates,[62] and a roof Gonioprism for use with the slit lamp that produces an erect image is reported by Colenbrander.[63]

The advantages of using transillumination, as well as direct illumination, with the Goldmann prism are mentioned by Dobree and Fincham,[64] and a simple device to facilitate its use is described.

8. *Fluorophotometry.*—A simple electronic apparatus which fits into a conventional Haag-Streit slit lamp and enables objective measurements to be made of the degree of fluorescence in the anterior chamber of the living eye is described by Langham and Wybar.[65]

D. Medical Treatment of Glaucoma

1. *Acetazoleamide* (Diamox).—One of the most exciting developments of the past year was the report of an entirely new type of drug for the control of elevated intraocular pressure. Acetazoleamide (Diamox; 2-acetylamino-1,3,4-thiadiazole-5-sulfonamide), a member of the sulfonamide family, has received considerable attention as a long-term diuretic, due to its inhibition of carbonic anhydrase. Its use in ophthalmology is based on Friedenwald's hypothesis of an active transfer of electrolytes into the eye by the ciliary body and Kinsey's findings of a remarkably high concentration of bicarbonate in the posterior chamber of the rabbit eye. The accumulation of the bicarbonate suggested that an enzyme such as carbonic anhydrase played an essential role in the formation of the aqueous humor, prompting Becker [66] to investigate the effect of this extremely potent carbonic anhydrase inhibitor on the intraocular pressure and dynamics. In almost all cases it has been able to reduce the intraocular pressure by its inhibition of the aqueous secretion.

Reports on its use by Grant and Trotter [67] and Posner [68] have also appeared in the literature. To date, all reports have been in agreement that acetazoleamide has little effect on normal eyes, its greatest effect being in secondary and acute narrow-angle glaucoma, where it often controls the pressure when miotics will not. In narrow-angle glaucoma its additional effect may be based on the reduction of the pressure differential between the posterior and the anterior chamber. In wide-angle glaucoma it usually lowers the pressure about one-third. It has few side-effects, the most annoying being the production of paresthesias, but it has not been used for a long enough period to show whether more serious effects will accompany its long-term usage. In the short period that acetazoleamide has been available, its use has been so successful that it seems to have established itself as an important member of our armamentarium against glaucoma.

2. *The Miotics.*—(a) General Considerations: In a series of three papers, Morrison ‡ discusses the neurohumoral theory of action of the miotics, their pharmacology, and the various factors affecting the transmission of the drugs. Finally, he studied the effect of cold, heat, and time on the stability and potency of pilocarpine, physostigmine, neostigmine, carbachol, and methacholine, and concluded that aqueous solutions of these miotics remained stable for as long as six months and resisted temperatures of 95 C. or freezing.

Tyner and Scheie [72] studied the frequently seen problem of the dilated, fixed, miotic-resistant pupil found in attacks of acute narrow-angle glaucoma. They concluded that the mydriasis seen with high intraocular pressure was independent of the preganglionic and postganglionic motor nerve supply of the iris sphincter of the dilator muscle and thus must be at the level of the sphincter muscle cells themselves. A critical level of increased pressure was found at which the pupil no longer responded to miotics and would not constrict unless the pressure was lowered by other means. This may be another factor explaining the occasional opening of the angle accomplished by acetazoleamide in miotic-resistant acute narrow-angle glaucoma.

(*b*) Cholinergic Drugs: (1) Pilocarpine. Lowenstein and Loewenfeld,[73] feeling that pupillometry was an ideal technique for the estimation of combined action

‡ References 69, 70, and 71.

of pilocarpine and physostigmine in the human eye, concluded that they were indeed additive and that their combined use was not contraindicated, as was suggested by Swan.

(2) Mintacol. The comparative strength of this new miotic was studied by Knupffer,[74] who felt that a 1:6,000 solution was equivalent to about the ordinary dose of pilocarpine. Caselli [75] showed that Mintacol (a phosphoric acid compound) produced a marked increase in the permeability of the blood-aqueous barrier 30 minutes after instillation into rabbit eyes.

(3) Physostine (trimethyl-furfurylammonium-*p*-toluene sulfate) was investigated by Rizzo,[76] who found that one drop of a 10% solution produced marked miosis, which started after 15 minutes and reached its maximum at about one hour. It lasted in good order for about 10 hours, necessitating administration of the drug only twice a day in most patients. It did not overcome atropine, and the writer noted no secondary effects.

(*b*) Anticholinesterase drugs: (1) Isofluorphate. The fact that isofluorphate (diisopropyl fluorophosphate; DFP) can produce an acute attack of glaucoma in eyes with a very shallow anterior chamber receives additional reinforcement by the report of two cases by Zekman and Snydacker.[77] The authors feel that isofluorphate is contraindicated in narrow-angle glaucoma.

Another untoward effect of isofluorphate is reported by Swan,[78] who since 1915 has found five patients with serious disturbances in vision due to obstruction of a miotic pupil by pigment cysts which developed after the administration of 0.1% isofluorphate. That this phenomenon is not found only with isofluorphate is shown in one case using carbachol and one using Mintacol.

3. *The Sympatholytic Drugs.*—Dibenamine caused a significant decrease in the rate of formation of aqueous in animals, according to DeLong and Scheie.[79] In air glaucoma it reduced the magnitude of the peak pressure, and in acute glaucoma significant reductions in intraocular pressure were found in three of five cases. It is the authors' feeling that the reduction in aqueous formation may be mediated by a blockage of epinephrine.

Hydergine, a mixture of equal parts of three hydrogenated ergot aklaloids, dihydroergocornine, dihydroergocristine, and dihydroergocryptine, was given to 27 patients suffering from different types of glaucoma by Agarwal,[80] with temporarily reduced tension in 19 patients. He concluded that Hydergine is principally of value in preoperative treatment and for the relief of pain in glaucoma.

Tetraethylammonium was found not to be a specific antiglaucomatous agent by Ljuština-Ivančić,[81] but it did demonstrate slight lowering of the ocular tension, especially in cases with coexistent cardiovascular hypertension.

4. *Miscellaneous Treatment.*—The analgesic effects of retrobulbar injections of tolazoline (Priscoline) in hemorrhagic glaucoma is reported by Stern.[82] This effect was lasting and was not related to normalization of the tension, which did not occur. A similar result of x-ray therapy is reported by Toniolo,[83] who feels that x-rays should be systematically tried before enucleation where tumor is not suspected. A total dose of 500 to 700 r in divided doses of 80 to 120 r was administered.

Corneal baths of 50% dextrose solution for 20 minutes were successful in lowering the intraocular pressure and clearing the cornea in primary glaucoma, but Löhlein [84] claims that in three patients with secondary glaucoma the tension was raised.

Ray,[85] Brown and Guilbert,[86] and Cascio and Lodato [87] reported the successful administration of the antispasmodics dicyclomine (Bentyl) hydrachloride and methantheline (Avagal) in glaucomatous patients.

E. Surgical Treatment of Glaucoma

1. *General Considerations.*—An interesting and plausible explanation for the acute glaucoma following sudden closure of the cyclodialysis cleft is reported by Goldmann.[88] The writer reports a case of an eye in which a cystic filtering scar developed after cataract extraction, a case of juvenile glaucoma, and a case of glaucoma simplex in which cyclodialysis had been done. All cases had gonioscopically open angles. When these new routes of aqueous outflow closed, all three eyes reacted with a rise of tension higher than the original tension. Because there were no gross anatomical disturbances in the Schlemm canal system, the writer concludes that new routes of aqueous outflow, with much lower resistance than that of Schlemm's canal, lead to a disuse obliteration of it.

Wyman,[89] in an experiment with rabbits, found that injecting subconjunctival cortisone after iris inclusion operations increased the amount of vascularization and fibrous tissue proliferation in the treated eyes. Unfortunately, this observation was not confirmed with histological sections.

2. *Iridectomy.*—Posner [90] and Barkan [91] discuss the extremely gratifying results of iridectomy in narrow-angle glaucoma. Barkan [92] then discusses the mechanism of iridectomy, which he feels is the neutralization of the higher pressure in the posterior chamber, resulting in a deeper anterior chamber.

3. *Iridencleisis.*—Lukić [93] has a new technique of iridencleisis which avoids the unfavorable effects of the change in the shape of the pupil, i. e., the appearance of the eye, and the spherical and chromatic aberrations in the lower parts of the visual fields. His technique consists of an oblique iridotomy, with preservation of the sphincter, and the incarceration of the tongue of the iris in the wound.

Marin-Amat [94] discusses the belief of the neurohumoral school that the iridencleisis works primarily by diminishing the trophism of the distended iris, acting as a sedative upon the plexus of Krause, localized in the insertion of the iris. He believes that any fistulization that is established between the anterior chamber and the subconjunctival spaces is not the principal, but merely the complementary, element of the therapeutic action of iridencleisis.

A seton operation in which an acrylic plate is substituted for the iris is reported by Qadeer [95] to have given good results in 14 eyes with primary glaucoma.

4. *Trephine and Anterior Sclerectomy.*—A new technique of opening the conjunctiva, by which the conjunctiva is pulled down to the limbus and only a very small buttonhole is made to admit the trephine, is reported by Verhoeff and Chandler.[96] It reportedly has the advantage of minimal dissection and rapid healing.

The mechanism and high incidence of iritis, with resultant anterior and posterior synechiae, during the postoperative course of trephine is discussed by Dudinoff.[97]

Trephine was successful in 80% of Rohrer's [98] cases but cyclodialysis in only 68%. However, Rohrer found that trephination increased the development of cataract. D'Ermo [99] states that he had good results with trephination in one old patient and iridectomy in another with chronic glaucoma and keratoconus.

The results of the "anterior flap sclerectomy with basal iridencleisis," first described in 1948 by Stallard,[100] are reviewed by its originator. This operation consists of a small, 5 mm., hinged anterior sclerectomy coupled with a small cyclodialysis and peripheral iridencleisis. Of 162 eyes with glaucoma it was successful in 97.9%, maintaining the pressure below 25 mm. The complications were few and of the ordinary type found with filtering operations.

5. *Cyclodiathermy.*—Weekers and Delmarcelle [101] studied the mechanism of cyclodiathermy with the fluorescein instillation technique and concluded that a very much reduced flow of aqueous was the result. Van Heuven and Dunn [102] studied histological sections in rabbits after cyclodiathermy and found an eventual scarring of the ciliary processes. Early, they found that the involved processes were necrotic and hemorrhagic and that the pigment layer was edematous. On the uninvolved side the ciliary processes were filled with blood and were edematous and hyperemic, and they felt that the acute rise in tension was due to this vascular reaction and stimulus.

The indications for cyclodiathermy are reviewed by Alajmo and Simonelli,[103] who found their best results in infantile glaucoma and glaucoma secondary to uveitis. Weekers and Delmarcelle [104] used it successfully against secondary glaucoma due to delayed formation of the anterior chamber following cataract extraction.

A large number of variations in technique are described. Velissaropoulos [105] tells of his experiences with Beren's cycloelectrolysis. Kamel [106] describes the perforating technique, the flat electrode, the subscleral coagulation of Thiel and Diaz Domingues, and the method of freezing the ciliary body with solid carbon dioxide. Splendid results (100%) with a combined cyclodiathermy and cyclodialysis were obtained in 54 eyes by Sokolić.[107]

Hurwitz [108] used cyclodiathermy as an office procedure in two cases, without complication.

6. *Cyclodialysis.*—Losche,[109] finding that cyclodialysis was not always effective, recommends combining it with iridectomy or iridodialysis. The idea of using a nonirritating plastic cannula in the cleft is entertained.

This reviewer, who has often wanted to close a cyclodalysis cleft, resulting in hypotony, found very interesting the unsuccessful attempts to do so in three cases described by Vannas and Bjorkenheim.[110]

7. *Goniotomy.*—During 17 years Barkan [111] normalized the pressure in 152 of 196 eyes with congenital glaucoma. He states that the operation succeeds by allowing the aqueous access to the trabeculum-canal-of-Schlemm mechanism. The operation of goniotomy is contraindicated in marked distention of the globe, but the author has has successful results in one case of aniridia and two cases of naevus flammeus. These results are confirmed by Paufique and Etienne,[112] who report 16 good results in 17 cases.

Stepanik [113] presents a combination of a cannula and a curette for use in goniotomy which has the advantage that fluid can be introduced into the anterior chamber during the operation in order to maintain its depth.

8. *Miscellaneous Operations.*—Filatoff and Kalfa [114] found detachment of the choroid in 57 of 230 patients operated on for glaucoma and concluded that the most characteristic feature in detachment of the choroid was the nonrestoration of the anterior chamber. If the chamber remains unrestored for many days, other complications set in—anterior synechiae and, later, dystrophy of the cornea. The

authors therefore attempted to hasten the restoration of the anterior chamber and the reattachment of the choroid by a posterior sclerectomy in cases in which the chamber was not restored in five to seven days. This operation leads to a decrease of tension in the posterior segment of the eyeball, and the accumulated fluid between the choroid and sclera is allowed to escape during the operation and the iris and the lens are pushed backwards; then the aqueous can flow freely into the anterior chamber.

Keyton [115] describes an operation which consists in incising the conjunctiva at a point between the insertion of the superior rectus and that of the external rectus. Tenon's capsule is then lifted and the sclera incised. So far, the author feels that his results have been satisfactory. The technique of opticociliary neurectomy for the relief of pain in absolute glaucoma is recommended by Basu.[116]

II. CONGENITAL GLAUCOMA

The pathogenesis of congenital glaucoma is characterized by François [117] as a persistence of embryonic mesodermal tissue in the iridocorneal angle; the absence, the malformation, or the malposition of Schlemm's canal, and a hypoplasia of the uvea, which are accompanied in varying degrees by an increase in tension and in dimensions of the globe. Payne,[118] after reviewing the histological sections of 37 eyes operated on for juvenile glaucoma, classifies the disease under two general types: (1) buphthalmos, in which the anterior chamber is compressed or absent as a result of broad adhesions of the iris to the corneal endothelium, and in which all medical and surgical procedures do poorly, and (2) hydrophthalmos, in which there is a deep anterior chamber, wide chamber angle, deficiency of the trabeculum, and possible absence of Schlemm's canal, a thin and atrophic iris, and small, partially dislocated lens. This type may respond to surgery, and the writer concludes cyclodialysis is better than iridectomy, sclerectomy, or trephining. Their experiences with trabeculectomy or cycloelectrolysis are too new for evaluation.

III. SECONDARY GLAUCOMA

A. Pseudoexfoliation of the lens capsule is a name coined by Dvorak-Theobald [119] to describe her extremely exciting contribution to the entity which most of us have mistakenly identified as capsular exfoliation. In a beautifully illustrated article, Theobald describes three cases of pseudoexfoliation characterized by accretions of an unknown material on the lens capsule, ciliary body, and zonules, and differentiates this condition from "true" exfoliation occurring in persons exposed to extreme heat over a long period of time. In the latter there is a rupture of the superficial layer of the capsule, which either becomes shredded in appearance or curls up in a scroll-like manner.

Pseudoexfoliation, on the other hand, is the disease we commonly refer to as glaucoma capsulocuticulare, and the accretions, of unknown etiology, are loosely attached to the iris, cornea, trabeculum, ciliary body, zonular fibers, and anterior limiting membrane of the vitreous. The debris invades the iris and fills the intratrabecular spaces, and even the canal of Schlemm, thus blocking all avenues of egress for the aqueous, and thereby producing a condition directly favoring an increase in intraocular pressure.

B. Pigmentary glaucoma of the Krukenberg spindle type is commoner than is generally believed, according to Calhoun,[120] who believes that corneal pigmentation of the Krukenberg type is an expression of a concomitant degenerative condition of the eye in which glaucoma may ensue, but who found no evidence that the pigment deposition was producing the glaucoma. As a point of practical value, he recommends the water-drinking test for early diagnosis.

C. Spontaneous rupture of the lens capsule as a cause of secondary glaucoma is characterized by the very acute cases in which the anterior chamber is suddenly flooded with liquefied lens cortex, which may resemble purulent endophthalmitis. A history of blindness due to cataract of several years' duration and a light gray color of the contents of the anterior chamber suggest rupture of the lens capsule. Hubbersty and Gourlay [121] suggest aspiration of the aqueous in order to see a rent in the capsule, and microscopic examination of the aqueous may show cholesterol crystals derived from degenerate lens cortex. The more chronic cases resemble cyclitis with secondary cataract. Deposits on the back of the cornea due to inflammation are sometimes very hard to distinguish from deposits of lens matter, and the diagnosis depends largely on finding a hole in the lens capsule.

D. Kirsch and Steinman [122] report a hitherto unreported form of secondary, postoperative glaucoma which responds to miotic therapy and is characterized by acute congestive glaucoma following diffuse rupture of the hyaloid membrane with generalized spread of the vitreous into the anterior chamber, blocking the angle.

Ross [123] reviews 22 cases of postoperative glaucoma in which the etiology could be traced to operative complications. In a similar vein, Wyman [124] reports two cases of postoperative glaucoma secondary to cataract extraction which he feels was due to the air he injected getting behind the iris and collapsing the chamber.

E. Glaucoma secondary to iridocyclitis is discussed by Weekers and associates.[125] These authors concur with Grant that the majority of cases of ocular hypertension in uveitis are due to an increased resistance to aqueous outflow. They believe that atropine and cortisone diminish the resistance to aqueous outflow and normalize the tension in the majority of cases of uveal hypertension of recent onset, but when these agents fail they recommend cyclodiathermy.

F. Miscellaneous forms. Bojer [126] tells of a case of iridoschisis in which acute glaucoma developed after the patient received a mydriatic. Posner and Schlossman [127] review their description of the syndrome of glaucomatocyclitic crises and differentiate this condition from acute narrow-angle glaucoma and glaucoma secondary to uveitis.

The glaucoma associated with epidemic dropsy has been linked to the contamination of cooking oils with an American weed, Argemone mexicana. L. Hakim [128] has produced acute increases in tension by the subconjunctival or intraocular injections of the active alkaloids of this weed and has sustained rises by the continued use of these alkaloids. His findings, chemical and pharmacological, and the ophthalmological evidence lend support to the theory of the toxic etiology of primary glaucoma in other parts of the world by indirect ingestion of papaver-like substances.

Finally, rare and interesting cases of glaucoma associated with systemic diseases are presented by Posner,[130] Riffenburgh,[129] and Raymond.[131]

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Correspondence

SOME ASPECTS OF TORSION

To the Editor:—In the June, 1954, issue of the ARCHIVES, page 783, is published an article by J. V. D. Quereau, M.D., entitled "Some Aspects of Torsion." This paper was read before the College of Physicians of Philadelphia, Section on Ophthalmology. In the July, 1954, issue of the ARCHIVES, page 166, there is a report of the discussion that followed. In this discussion the Editor of the ARCHIVES participated.

This letter is in no manner intended to be derogatory to or hypercritical of this paper or of the discussion reported. There are, however, some pertinent facts that should have been included in a paper of this nature. Also, I should like to raise my voice in protest against some of the conclusions found both in the original article and in the report of the discussion that followed. Possibly this protest will be more lucid if put in question form.

In his article, Dr. Quereau states (page 785): "To observe accurately rotations of the eyeball, we must surround the eye with a spherical system of coordinates." Dr. Quereau then describes his technique for the objective determination of what he terms real torsion. Subsequently, Dr. Quereau says, "This will be real torsion, for false torsion is entirely eliminated from the picture."

In the July, 1954, issue of the ARCHIVES (page 166) Dr. Adler states, "Dr. Quereau has very clearly defined what false is and how it compares with what we call true torsion. One might say that Dr. Quereau has laid the ghost of true torsion in the oblique positions of gaze, and that it would take somebody with considerable temerity to try to disinter it."

MATTERS CONSIDERED PERTINENT

In 1847, Donders, after discovering that torsion of the globe was dependent upon the direction or position of gaze, formulated his law, which has been repeated in many texts and publications. It is noteworthy that in this law Donders referred to this torsion *with respect to the head*. Donders did not state that all eyes had the same angle of torsion for an identical position of gaze. Donders did not state that all of the many variations of an oblique position were accompanied by a plus or minus angle of torsion. Donders did not state that when the direction of gaze was directly up or down, or directly to the right or the left, this angle of torsion was necessarily zero. However, in regard to the last two statements, I would interject that I believe that relatively, and for all practical purposes, these statements are true, provided that one is dealing with an eye that has no neuromuscular abnormality.

In substance, Donders' law asserts that the angle of torsion with respect to the head is controlled solely by the position of gaze. Helmholtz confirmed the findings of Donders and gave a law expressed in different terms (Duke-Elder, S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1932, Vol. I, p. 596).

In the 100 and some odd years that have followed the statement of Donders' law, some investigators of torsion have made conclusions that are contrary to those of Donders. In short, these investigators thought they had proved that an independent torsional movement or cycloduction occurred as a normal function of the

eye. In general, these deductions have been accepted as being entirely accurate, although there is little or no mention in texts of these deductions being contradictory to those of Donders. I believe that herein originates much of the confusion and perplexity that concerns torsion. Actually, a pure torsional movement, independent torsional movement, cycloduction, and cyclophoria are all contradictory to Donders' law. They are by definition incompatible.

It appears that several years ago one writer, in preparing a text on ocular motility, must have recognized these contradictory concepts and felt that Donders and Helmholtz had made erroneous deductions. The term false torsion was applied to the torsion described by Donders. I would add that this most unfortunate term (false torsion) must have arisen without adequate investigation as to which concept was correct and that little attention was given to the two being contradictory. Possibly this was done in a spirit of being gracious to Donders. I do not know. Nor am I certain as to the exact meaning this term is intended to convey. However, I believe that it can be stated that this term did not alleviate or diminish the confusion about torsion.

A few years ago, recognizing these contradictions, I made an effort to determine which of these concepts was correct. Experiments were conducted to prove or disprove the validity of one of the contradictory concepts. A study of the reports indicating the existence of cycloduction was made. In this investigation I found (1) nothing contradictory to Donders' law but did find confirmatory evidence that the angle of torsion was altered by a change in the position of gaze, and (2) what I consider absolute proof that a head tilt toward the shoulder did not cause or result in an independent or a reflex torsional movement, as is currently accepted. These findings were reported in an article entitled "Torsional Eye Movements" (*Am. J. Ophth.* **34**:253 [Feb.] 1951).

In this paper reference was also made (possibly in too mild a manner) to the reason why false conclusions concerning the validity of cycloduction or an independent torsional movement had resulted. After a study of these papers, the answer to me was obvious. It was not in faulty observations or recordings but, rather, in the failure to recognize or to bear in mind the full significance of Donders' law.

I have no doubt that when the complete story of torsion is finally obtained, it will be found that cycloduction, cyclophoria, independent torsional movement, pure torsional movement, and reflex torsional movement are the real false culprits. They are the impostors.

It is a challenge to ophthalmology to determine conclusively which of these concepts is true and then to bar the impostor, or false concept, from being treated as authentic in future authoritative texts dealing with ocular motility. This challenge can and should be met with tact and fortitude.

COMMENTS AND QUESTIONS

The comment and questions that follow immediately are addressed especially to Dr. Quereau.

In your paper you create a new definition, or at least a new standard, for the determination of real torsion.

1. Is it not quite possible that if this new definition or standard is accepted as authentic, it will only add to the confusion that concerns torsion?

2. Do you have any evidence from your investigation that an independent or a pure torsional movement occurs as a normal function of the eye?

3. Is the real torsion that you describe contradictory in any manner to Donders' law?

4. Do you consider the real torsion that you describe to be a type different from that described by Donders? If the two types are not quite identical, is not yours a subclassification of the Donders type?

5. Would you give a clear, concise, and understandable definition of your understanding of false torsion?

The comment and question which follow are addressed especially to Dr. Adler.

I believe that clarification of torsion opens the front gate, so to speak, to increased practical knowledge of ocular motility, orthoptics, refraction, and possibly other related branches of medicine. Therefore, I consider this new definition or standard of much importance. I also believe that any scientific article appearing in a journal of the caliber of the ARCHIVES conveys some sense of authenticity, even if this is of a limited nature; and when this new definition is completely endorsed by the Editor, even if not as an editorial, this sense of authenticity is definitely augmented. My question follows: Before this new definition or standard for the determination of real torsion becomes fully accepted, would it not be better to have this matter considered by a committee or body representing ophthalmology in this country, or even internationally?

Actually, there are other matters in the discussion, especially concerning reciprocal innervation and lines of space, that I should like to delve into, but I fear this letter is much too long now.

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To the Editor:—The following is a reply to the questions asked by Dr. Hewitt.

Question 1.—Is it not quite possible that if this new definition or standard is accepted as authentic, it will only add to the confusion that concerns torsion?

Answer: This certainly is possible. On the other hand, any new light on the nature of false or apparent torsion and the means by which false torsion may be avoided when observing real torsion should be helpful.

Question 2.—Do you have any evidence from your investigation that an independent or a pure torsional movement occurs as a normal function of the eye?

Answer: Yes. When the eye moves from a secondary or tertiary position to any other secondary or tertiary position, it undergoes real torsion. This includes nearly all movements of the normal eye, since the eye rarely begins its motion exactly from the primary position. The means by which this torsion is produced is described by Duke-Elder (Text-Book of Ophthalmology, Vol. I, pp. 599-601). A new point of view has developed on this subject as the result of using the spherical system of coordinates in observing rotations of the normal eye. The meridians of longitude used in this system fan out from the primary position. Like the meridians of longitude from the North Pole of the earth, they run backward and meet again behind the eye at a "south pole." As the eye rotates in any direction from the primary position to a secondary or tertiary position, it follows one of these lines of longitude, and the meridians of the cornea maintain a constant angle with the line of longitude which the cornea is following. The vertical meridian of the cornea maintains the same angle with this meridian of longitude as it did when the eye was in the primary position, before the rotation was begun. Therefore, no torsion has occurred, because true torsion results in a change in this angle. The eye rotates in such a manner that it maintains a "no-torsion" position in relation to the meridian along which it is moving.

Suppose the left eye has moved upward and outward about 30 degrees. Suppose that from this position it next makes a rotation straight downward to a position 30 degrees downward and outward. In doing this, the cornea crosses many meridians of longitude (an infinite number), and as it crosses each meridian of longitude the vertical meridian of the cornea assumes the same angle it would make with this meridian of longitude if the cornea were in the primary position. Paradoxically, to assume this no-torsion position in passing from one meridian of longitude to another

requires an act of real torsion. Anyone can prove this. Take a globe map of the earth and turn it on its side so that the north-south axis faces the observer and is horizontal. Hold a fountain pen tangent at the North Pole to represent the vertical meridian of the cornea in the primary position. Move the pen, still vertical, to the right as far as the equator. The pen has maintained its angle of 90 degrees with the horizontal meridian of longitude, and when it arrives at the equator it is parallel to the equator. Starting again from the primary position, move the pen along the 45-degree meridian, with which the pen held vertically in the primary position makes an angle of 45 degrees. Maintain this 45-degree angle, the no-torsion angle, with the meridian of longitude until the pen crosses the equator. It will cross the equator at 45 degrees to the equator; any other angle would mean that the eye had undergone real torsion in its movement from the primary position. (This constant angle can easily be observed by means of a linear after-image moved along the edge of a perimeter arm.) If the eye is now moved from this second position, in which the vertical meridian is at 45 degrees with the equator, down to the first position, in which the vertical meridian of the cornea is parallel with the equator, it is obvious that the eye must undergo 45 degrees of real torsion, all to preserve the no-torsion relation between the corneal meridians and the meridians of longitude which the cornea is crossing. This corrective, real torsion is produced by rotation of the eye around an axis which lies in a plane, the secondary axis plane, situated halfway between Listing's plane and the equator of the eye. It is described by Duke-Elder (Text-Book of Ophthalmology, Vol. I, pp. 599-601). All rotations of the normal eye which do not move the cornea directly toward or away from the primary position are associated with real torsion. Probably all rotations of the normal eye when the visual lines are parallel should be said to take place around an axis which lies in the secondary axis plane. In the primary position this plane coincides with Listing's plane. It is unfortunate this was not called Listing's plane, since very few rotations take place around axes lying in the plane which now bears his name.

To the subject of real torsion with head tilt, with convergence and with upward and downward gaze during convergence, I have nothing new to add.

Question 3.—Is the real torsion you describe contradictory in any manner to Donders' law?

Question 4.—Do you consider the real torsion that you describe to be a type different from that described by Donders? If the two types are not quite identical, is not yours a subclassification of the Donders type?

Answer: These questions can be answered as one. The real torsion I describe is not contradictory to Donders' law. Donders and Listing knew that the "torsion" they described was not a rotation around the line of fixation. If only Donders had said: "For any determinate position of the line of fixation with respect to the head, there corresponds a definite and invariable angle of *apparent* torsion," all the confusion would have been avoided, for torsion in America has come to mean rotation around the fixation axis, i. e., real torsion. We speak of intorsion in extraocular muscle paresis or describe a certain muscle as an extorter; actually, these movements should be called rolling, and the appearance of rolling which occurs when the eye moves obliquely from the primary to a tertiary position, and which is only a false appearance of rolling and is not a turning around the line of fixation, should be called torsion. This difference is described by Duke-Elder (Vol. I, p. 598).

The real torsion I described is a true rolling around the fixation axis, and rolling would be a better term for it. The torsion which Donders describes is not a rolling around the fixation axis, but it gives this appearance. There is no conflict between them. They are two different entities. The illusion of rolling—torsion—which Donders describes complicates the objective detection of actual rolling—real torsion—but if the spherical system of coordinates is used when looking for rolling,

the illusion of rolling, Donders' torsion, will not be seen. It is easy to employ the spherical system of coordinates in observing eye movements. All that is necessary is to look at a corneal meridian which lies in the direction of the proposed rotation of the eye and to sight along this corneal meridian and direction of rotation. Real torsion, rolling, if any, can then be seen without the illusion of rolling to mask it.

Question 5.—Would you give a clear, concise, and understandable definition of your understanding of false torsion?

Answer: False torsion is the angle between the vertical meridian of the cornea (or retina) and a vertical plane which passes through the center of rotation of the eye and the fixation point. This angle, erroneously interpreted as rolling around the visual axis, is merely the result of using a planar or rectangular coordinate system, instead of a spherical coordinate system, to locate surface markings on a rotating sphere. This illusion of rolling is called "false torsion," but "torsion" should be sufficient.

The spherical coordinate system provides a different point of view, in which a corneal or retinal meridian which has undergone 90 degrees of torsion in respect to the planar system has not undergone any torsion in respect to the spherical coordinate system.

J. V. D. QUEREAU, M.D.
138 N. 5th St.
Reading, Pa.

In a certain sense, Dr. Hewitt's remark that publication of an article conveys some sense of authenticity is correct, but this does not mean that the opinions expressed in the article are necessarily those of the Editorial Board. If the Editorial Board published only those articles with which it agreed, much new and valuable material would be omitted. The ARCHIVES OF OPHTHALMOLOGY tries to publish only those articles which present reasonable documentation of the author's conclusions, regardless of whether or not the Editorial Board agrees with them. In such a controversial subject as torsion it would be very unlikely to find any unanimity of opinion among the members of the Editorial Board, nor do I believe much would be gained at the present time in forming a committee to decide this matter ex cathedra. What we need is more experimental data to give us a correct answer to these perplexing problems.

FRANCIS HEED ADLER, M.D.

PREPLACED CORNEOSCLERAL SUTURE

To the Editor:—I was interested in a clinical note by Dr. Abraham J. Lanchner entitled "A New Preplaced Corneoscleral Suture," which appeared in the July 1954 issue of the ARCHIVES, page 129. An almost identical suture has been routinely used in the Department of Eye Diseases of this college since the beginning of 1953 and has been described elsewhere.[1]

The preplaced track suture, which suggested itself to me in 1952, after I had observed the postplaced track sutures commonly employed by the surgeons in the Massachusetts Eye and Ear Infirmary, Boston, has now been used in nearly four hundred operations here. As a result of the experience gained, we have shifted the position of the track needle a little posteriorly, so that its corneal end emerges at the limbus, giving a little larger section and an easier approach to the root of the iris.

During the course of development of this stitch we have tried to cut on the suture needle, but in a number of cases the suture became frayed in an attempt to pick it up with a lens hook.

MAHMUD ALI SHAH, M.B., M.S.
Dow Medical College
Karachi, Pakistan

REFERENCE

1. Shah, M. A.: The Corneo-Scleral Track Suture: A New Technique, Brit. J. Ophth. **38**:239, 1954.

TRACHOMA

To the Editor:—Charles Snyder, in his report on the "First International Congress of Ophthalmology and Contemporary State of Ophthalmology in America," published in the August, 1954, issue of the ARCHIVES, page 264, describes the attention given at that time (1857) to trachoma, which "had been the scourge of the armies of Europe for more than a half a century." In that report, transmission, treatment, and prevention were discussed. Any discussion of the problem today would proceed on similar lines. Trachoma, as a contagious disease, became, following the practice of the Belgian General Staff, a valid reason for excluding affected persons from the draft.

In 1919 and 1921, while still in Europe, I saw and treated several men suffering from trachoma. These men had fled from the Bolshevik revolution in Russia. These patients volunteered the information that they had infected themselves with trachoma deliberately, in order to evade induction into the Russian Czarist army. These patients stated that they had used fresh, contagious smear from the eyes of persons who had active trachoma. They had, of course, never undergone any treatment for the condition, so long as they had stayed on Russian soil. All of them had a progressive pannus covering both corneas and, accordingly, very reduced visual function.

At the time of this voluntary and intended self-infection, nobody, in an endemic area, could have suspected, or been sure of, the horrible origin of this "transmission."

WERNER BAB, M.D.
350 Post St.
San Francisco 8

NOTCHING OF OPTIC CHIASM BY OVERLYING ARTERIES IN PITUITARY TUMORS

To the Editor:—In their article entitled "Notching of the Optic Chiasm by Overlying Arteries in Pituitary Tumors," published in the February, 1954, issue of the ARCHIVES, pages 161-170, Rucker and Kernohan presented five cases of pituitary tumors with damage to the chiasm by the circle of Willis. They quoted L. Türck as the first author who, 100 years ago, described the strangulation of the optic nerves by the circle of Willis. Rucker and Kernohan, after having cited Türck, skipped all other publications until that of Fay and Grant in 1923. They cited, further, a candidate thesis by Lillie, 1931, and studies of Balado and Malbrán, 1933, and they gave two other references at the end of the text.

Rucker and Kernohan overlooked my article "Symptoms and Treatment of Pituitary Tumors," published in the *A. M. A. Archives of Otolaryngology* (**55**:268-306, 1952). They would have found there three very instructive pictures of the notching of the chiasm by the anterior communicating artery. And if they had read or turned the pages of T. Fay and F. C. Grant's publication in the *Archives of Neurology and Psychiatry* (**9**:739-750, 1923), they would have found on page 745 two large copies of my pictures, with the legend "(copied from the report by Hirsch [1921])." In my article in the *A. M. A. Archives of Otolaryngology* in 1952 can be found references to other publications in which I tried to convey to ophthalmologists knowledge of the role of the circle of Willis in the strangulation of the chiasm and the explanation of the mechanism causing bitemporal hemianopsia.

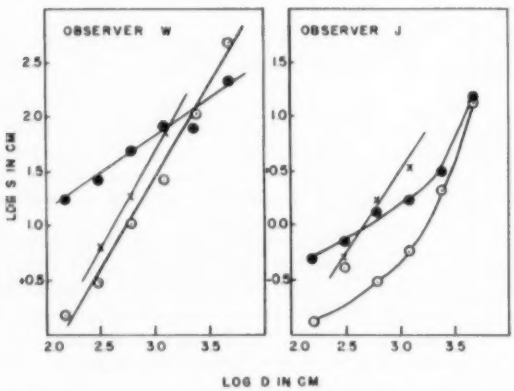
It may be of interest that Türck's publication was forgotten even in his native city of Vienna. When the subject of temporal and bitemporal hemianopsia was discussed in the Ophthalmological Society of Vienna in 1904 (*Ztschr. Augenh.* **13**:70, 1905), none of the ophthalmologists and neurologists present referred to Türck's publication. But Türck's specimen was at that time still in the Rokitansky

Museum of the Department of Pathology in Vienna. Erdheim, who became interested in pituitary tumors, saw it there with a number of other specimens of pituitary tumors. In one of the following meetings of the Ophthalmological Society of Vienna (*Ztschr. Augenh.* **13**:376, 1905), Erdheim demonstrated 12 specimens of pituitary tumors showing the strangulation of the optic nerves by the circle of Willis. I know these specimens, and therefore I was able to cite Türck's merit, as Erdheim did.

But I should like to point out that Türck did not use his observation to explain temporal resp. bitemporal hemianopsia. At that time the semidecussation of the optic nerves were not known. This explanation can be attributed to both Erdheim and myself.

Oscar Hirsch, M.D.
186 Commonwealth Ave.
Boston 16

CORRECTION



The graph published as Figure 2 (p. 528) of the article "Factors Involved in Several Tests of Binocular Depth Perception," by Louise L. Sloan, Ph.D., and Adelaide Altman, A.B., in the October issue of the Archives should be replaced by the graph above. The legend in the Archives is correct.

Obituaries

IGNATIUS GEORGE SOMMERS, M.D.
1894-1954

Ignatius G. Sommers died of lymphoma on Aug. 18, 1954, at the age of 60, after a month's illness. He was born in 1894 in Vienna, Austria, where he attended the Medical School of the University of Vienna, from which he graduated in 1919 with the degree of Doctor of Medicine.

Dr. Sommers served as intern and resident at the Vienna General Hospital and at the Vienna Polyclinic Hospital from 1919 to 1925, places and times in which shone some of Europe's brightest medical lights. The major portion of this time was spent in the departments of ophthalmology and otolaryngology, and the lesser portion in general surgery, internal medicine, dermatology, neurology, and pathology. From 1925 to 1927 he was assistant to Professor Sachs in ophthalmology, and from 1927 to 1932 he was assistant to Professor Alexander in otolaryngology.

After his residency training he made several lecture trips from Austria to the United States. While on one of these trips, in 1929, he received license by examination in Illinois. Subsequently, he was licensed in New York, New Jersey, and California. In 1938 he made the United States his home. In 1944 he acquired one of his most prized possessions, that of naturalized American citizenship.

From 1939 through 1942 he was lecturer in ophthalmology at the Long Island College of Medicine, in Brooklyn, and attending pathologist at the Harlem Eye and Ear Hospital in New York. In 1943 he moved to Chicago and became a fellow in pathology at the Cook County Hospital. From 1943 through 1945 he was surgeon and pathologist at the Chicago Eye, Ear, Nose and Throat Hospital.

In 1947 Dr. Sommers moved to Los Angeles, where he went into private practice in ophthalmology and otorhinolaryngology. In 1948 he became consultant in diseases of the eye, ear, nose, and throat to the United States Public Health Service in Los Angeles and San Pedro. In 1949 he was appointed Assistant Professor of Ophthalmology at the College of Medical Evangelists.

He was a fellow of the American Academy of Ophthalmology and Otolaryngology and of the American Medical Association, and was certified by both the American Board of Ophthalmology and the American Board of Otolaryngology.

Beginning in 1926, he gave numerous graduate and postgraduate courses in Vienna, New York, Chicago, and Los Angeles. The subjects included ophthalmology, otolaryngology, pathology, and neurology. The same wide range of subjects was covered in his amazingly prolific publications. His contributions to medical periodicals number one hundred six and were written in English, German, and French. He was co-author with Spiegel of a widely used textbook, "Neurology of the Eye, Ear, Nose and Throat," and author of another text, "Histology and Histopathology of the Eye and Its Adnexa." Both volumes are classics in their fields and were published in English, German, and Spanish.

Dr. Sommers' many students are to be found all over the world. He imparted to them not only of his vast storehouse of medical lore, filtered and distilled into

crystal-clear nuggets of descriptive phrases, but also of his kindness and patience, and of his sparse, but sparkling humor, which shone warmly through his extreme modesty and humility. He was fluently familiar with the Greek, Latin, English, German, Spanish, and French histories, mythologies, languages, and literature, and he often spiced his lectures with digressions into these cosmopolitan fields to tie into the medical topic under discussion.

He was guided in his personal and professional life by his motto: "Accumulate wisdom, not wealth." To maintain that ideal after him, he willed his body, his microscope, and his vast collection of histological and pathological slides for the teaching of oncoming medical doctors.

His wife, Vita S. Sommers, Ph.D., survives him and carries on their mutual interest in medicine in her daily vocation as a clinical psychologist caring for United States Service veterans.

Dr. Sommers' friends and colleagues deeply deplore his untimely death. Too early an eminent career has ended, and medicine has lost one of its most industrious and dedicated leaders.

VICTOR M. MARSHALL, M.D.

News and Comment

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Pan-American Congress.—The Fifth Pan-American Congress of Ophthalmology will be held in Santiago, Chile, Jan. 9 to 14, 1955. The official topics, and the moderators and discussers, are as follows:

1. Collagen Diseases	Moderator: Dr. John McLean Discussers: Drs. Bruce, Leopold, and Contardo
2. Glaucoma	Moderator: Dr. Peter C. Kronfeld Discussers: Drs. Friedenwald, Sugar, Grant, Barrenechea, Rocha, Belfort Mattos, and Antón
3. Congenital Glaucoma	Moderator: Dr. Edward Maumenee Discussers: Drs. Agüero, Tosi, Manzitti, and Bertotto
4. Secondary Glaucoma	Moderator: Dr. Jorge Valdeavellano Discussers: Drs. Rocha, de Rezende, Shahan, Trujillo, de Arruda, Cordero Moreno
5. Strabismus	Moderator: Dr. James H. Allen
6. Detachment of the Retina	Moderators: Dr. Juan Verdaguer Dr. Humberto Escapini Discussers: Drs. Teng, Schepens, Millán, McDonald, Picó, Swan
7. Psychosomatic Ophthalmology	Moderator: Dr. Daniel Snydacker
8. Tropical Diseases	Moderator: Dr. Durval Prado
9. Physiopathology and Surgery of the Crystalline Lens	Moderator: Dr. Derrick Vail Discussers: Drs. Schoh, Silva, Hortsmann, Haro, Barrenechea, and Espildora
10. Plastic Surgery	Moderator: Dr. Alberto Gormáz Discussers: Drs. Callahan, Spaeth, Hughes, Paiva, Paton, and Cutler
11. Visual Fields and Neuro-Ophthalmology	Moderators: Drs. Magín Diez, Oribe, and Adrogue Discussers: Drs. Grauer, Henderson
12. Intraocular Tumors	Moderator: Dr. Algernon B. Reese Discussers: Drs. Locke, McGavic, AraujoAzambuja, Benedict

Attention is called to the fact that Chile has a very pleasant climate, with beautiful scenery, magnificent hotels, and great natural resources. The Universidad de Concepcion is considered one of the best universities in the world. The Committee on Organization invites you to the Congress. Your presence will aid in the success of the Congress and in the reunion of Pan-American solidarity.

DR. RENE CONTARDO
Huerfanos 930, Of. 74
Santiago de Chile, South America

Stanford Spring Postgraduate Conference in Ophthalmology.—Stanford University School of Medicine will present the annual Postgraduate Conference in Ophthalmology from March 21 through March 25, 1955. Registration will be open to physicians who limit their practice to the treatment of diseases of the eye; or the eye, ear, nose, and throat. In order to allow free discussion by members of the conference, registration will be limited to 30 physicians.

Instructors will be Dr. A. Edward Maumenee, Dr. Dohrmann K. Pischel, Dr. Jerome W. Bettman, Dr. Max Fine, Dr. Earle H. McBain, and Dr. Arthur J. Jampolsky.

Programs and further information may be obtained from the Office of the Dean, Stanford University School of Medicine, 2398 Sacramento St., San Franscico 15.

University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology.—The Ninth Annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach the week of Jan. 17, 1954. The lectures on ophthalmology will be presented on Jan. 17, 18, and 19, and those on otolaryngology on Jan. 20, 21, and 22. A midweek feature will be the Midwinter Convention of the Florida Society of Ophthalmology and Otolaryngology on Wednesday afternoon, Jan. 19, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at 8 p. m.

The Seminar lecturers on ophthalmology this year are Dr. William F. Hughes Jr., Chicago; Dr. Phillips Thygeson, San Jose, Calif.; Dr. James Allen, New Orleans; Dr. Walter H. Fink, Minneapolis, and Dr. Milton L. Berliner, New York. Those lecturing on otolaryngology are Dr. Paul Holinger, Chicago; Dr. Lawrence R. Boies, Minneapolis; Dr. Edmund P. Fowler Jr., New York; Dr. Arthur W. Proetz, St. Louis, and Dr. David D. DeWeese, Portland, Ore.

Annual Clinical Conference Wills Eye Hospital.—The Seventh Annual Clinical Conference of the Staff and Ex-Residents of Wills Eye Hospital will take place at the hospital, 1601 Spring Garden St., Philadelphia 30, on Feb. 18 and 19, 1955.

Algernon B. Reese, of New York, will deliver the Arthur J. Bedell Lecture, entitled "The Diagnosis and Treatment of Orbital Tumors and Simulating Lesions."

SOCIETY NEWS

St. Louis Ophthalmological Society.—The St. Louis Ophthalmological Society held its first program of the year on Oct. 28, 1954, at the Park Plaza Hotel. The Washington University Department of Ophthalmology conducted the clinical portion of the program.

A symposium on "Glaucoma: New Concepts" was presented by Dr. H. R. Hildreth, Dr. R. D. Mattis, Dr. B. Y. Alvis, Dr. H. P. Venable, and Dr. B. Becker.

The regular meetings for this fiscal year will be held on the following Thursdays: Dec. 2, 1954; Jan. 27, 1955; Feb. 24, 1955; March 24, 1955, and April 28, 1955.

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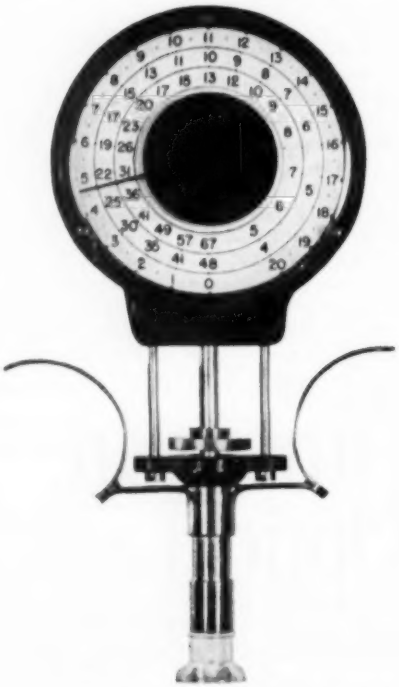

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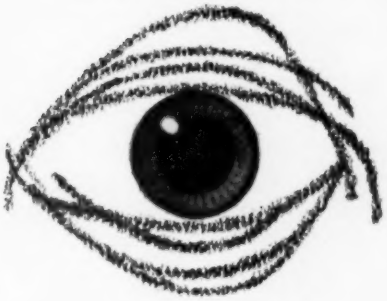

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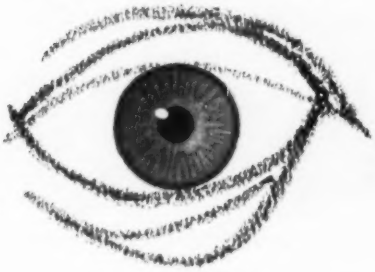

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1. Gettes, B. C., and Leopold, I. H.: A. M. A. Arch. of Ophth. 49:24 (Jan.) 1953.
2. Stolzar, I. H.: Am. J. Ophth. 36:110 (Jan.) 1953.

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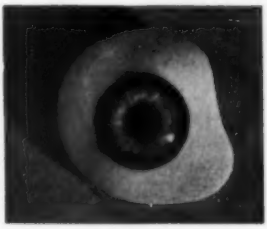

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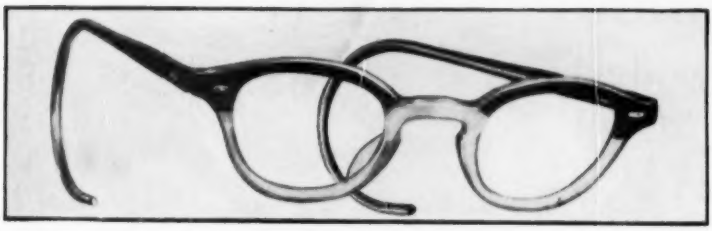

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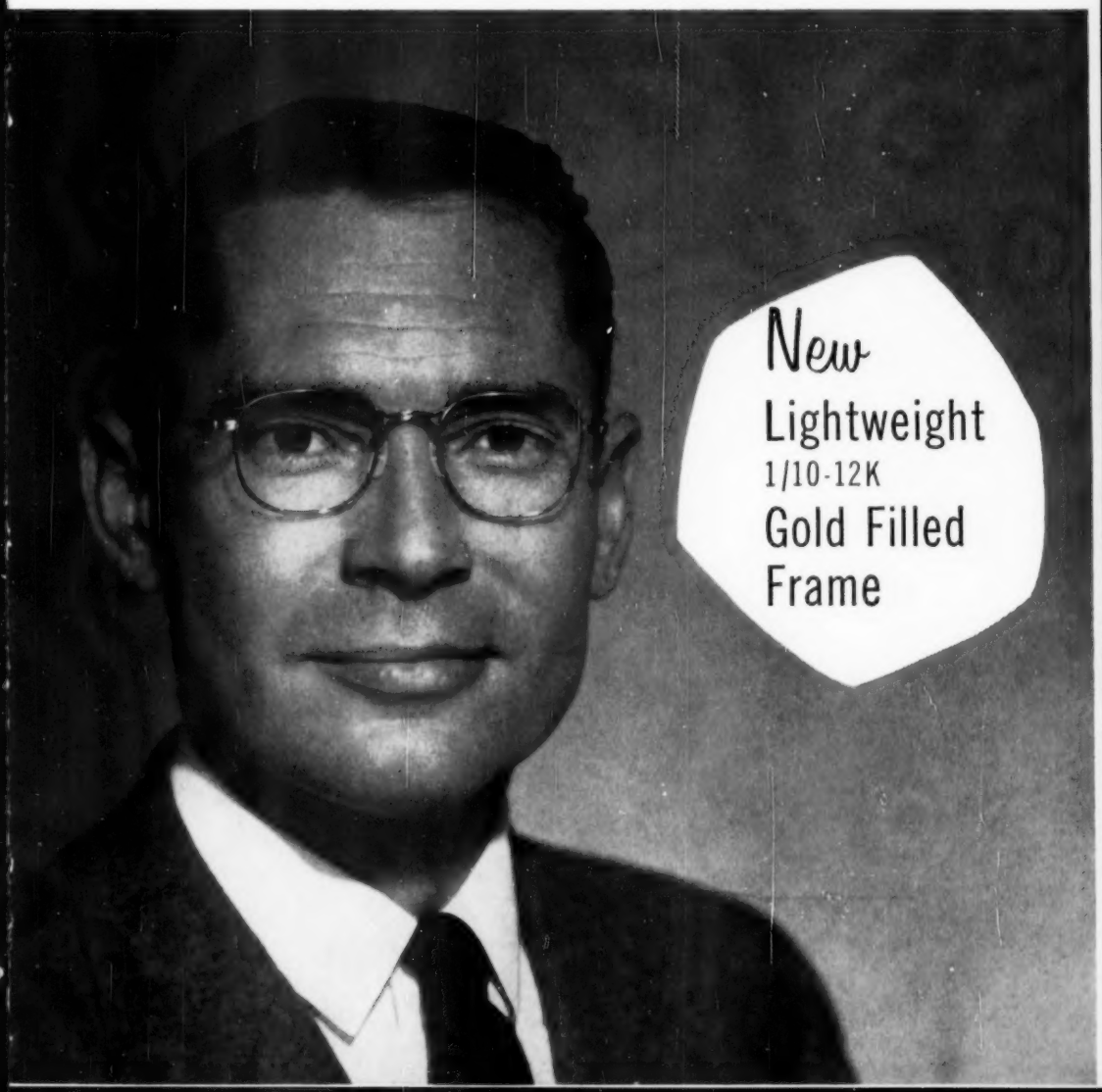

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